The New England
Journal of Medicine

VOLUME 209 SEPTEMBER 21, 1933 NUMBER 12

Massachusetts Medical Society

SECTION OF SURGERY

Tuesday, June 6, 1933

PRESIDING:

Dr. Allen G. Rice, Springfield, Chairman.
Dr. Ernest M. Daland, Boston, Secretary.

CHAIRMAN RICE: The meeting will come to order. Your committee, quite encouraged by the success of last year's program which took up a subject of general interest, concerning which there was considerable, favorable comment, has arranged a similar program for this year, this time on hernia. The first paper is by one who has come to understand that this navel ill may be fraught with peril.

Dr. Richard H. Miller, of Boston.

UMBILICAL HERNIA*

BY RICHARD H. MILLER, M.D.,† AND MARSHALL K. BARTLETT, M.D.†

THE purpose of this presentation is to call again to the attention of the medical profession the potential danger of large umbilical hernias in adults. Physicians and surgeons alike have been too prone to look with complacency on this condition, and too ready to advocate palliative treatment, with belts and supports, for a type of lesion which has inherent possibilities of grave sequelae.

A scrutiny of the literature does not reveal many writings on the subject of umbilical hernia. W. J. Mayo in 1899[9], and again in 1903[10], described his method of transverse incision, with overlapping closure of the opening, a technique now almost universally carried out. Simmons[12], in 1916, reported 70 operative cases from the Massachusetts General Hospital and found the results of the Mayo operation better than those of any other procedure, stating that recurrences following other methods could be expected in 46.4 per cent. Cullen[3], in his book on Diseases of the Umbilicus, in 1916, reviewed the subject carefully. Goldman[6] discussed this condition in 1917, and Delatour[4] in 1918. Warren[13] presented statistics of 49 cases, in 1920, which bear out the thesis of this paper, that operation is safer than expectant treatment. His figures were as follows:

	Operations	Mortality
Simple hernia repair (non-strangulated)	11	0%
Strangulated hernia	32	25%
Strangulated hernia (repair with intestinal resection)	6	33%

*From the Clinic of the Massachusetts General Hospital.
Read before the Section of Surgery of the Massachusetts Medical Society at its Annual Meeting, June 6, 1933.

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Foss[5] recommended free intestinal resection in large hernias with adherent irreducible bowel. Cullen[2] and Roeder[11], and Counseller and McIndoe[1], have written on the operative technique. La Roque[7] has described his method for closing large defects with patches of fascia lata. The technique of operation is of moment, but of far greater importance is it for the surgeon to realize the value and even the necessity of operation in simple non-strangulated cases, because every patient with a large umbilical hernia is courting disaster.

At birth there are two types of umbilical hernia which are serious and require immediate attention, but both are fortunately rare. The first is hernia into the cord, close to the abdominal wall; there is danger, in this event, of tying the tape around the intestine itself, if the bowel protrudes far enough and if the suture is placed too close to the abdomen. The second is the so-called amniotic hernia, in which the skin and underlying tissues of the anterior wall are, for a space, lacking, and the abdominal contents are here covered and protected only by amniotic membrane. Where this condition obtains, immediate operation is imperative.

The common hernia of infancy is the simple bulge at the umbilicus, easily reduced, and usually cured, with adhesive plaster. This simple procedure suffices to cure most cases, but if they persist, enlarge or cause symptoms, they may at any time be repaired by very simple operative measures.

In adults a simple bulge at the umbilicus, small and round and easily reduced, is not of serious significance, and so long as it does not increase in size it does not, per se, necessarily indicate surgical attention.

The large irregular umbilical hernia, more common in adult females, containing bowel which is often adherent, or reducible with difficulty, is the type which constitutes a serious menace. In 1931 we reported, at the meeting of the New England Surgical Society, 349 cases of umbilical hernia operated on at the Massachusetts General Hospital in the 20 years from 1911 to 1930, inclusive[8]. Of these 349 cases, 289 (83%) were simple, and 60 (17%) were strangulated. In the 289 operations for simple hernia there were seven deaths, a mortality of 2.4%, whereas in the 60 strangulated cases there were 18 deaths, a mortality of 30%.

*TABLE I

UMBILICAL HERNIA

1911-1930

Total Cases 349

Simple	289	(83%)	Strangulated	60	(17%)
Deaths	7		Deaths	18	
Mortality	2.5%		Mortality	30%	

*This table taken from a previous paper.

In the 10 years from 1921 to 1930, inclusive, there were 185 operations for simple hernia with six deaths (3%) and 38 operations for strangulated hernia with 12 deaths (31%). These statistics demonstrated that in a large urban hospital we found in 20 years about 350 cases of umbilical hernia, with seventeen per cent of them strangulated, and whereas the mortality for operation on the simple type was three per cent or under, for strangulation it was thirty per cent.

TABLE II

UMBILICAL HERNIA

1920-1930

Total Cases 233

Simple	185	(83%)	Strangulated	38	(17%)
Deaths	6		Deaths	12	
Mortality	3%		Mortality	31%	

TABLE III

STRANGULATED UMBILICAL HERNIA

38 Cases

Males	9 (31%)
Females	29 (69%)
Average age	59
1 case aet.	2
(38 Cases)	

A careful review of the cases admitted to the same hospital in the years 1931 and 1932 only serves to give force to our contention, and the figures, though few, are still more striking than those previously recorded. The diagnosis of umbilical hernia was made in 34 instances, in 12 of which the finding was incidental, leaving 22 cases in which the hernia was the major cause of hospitalization.

Of these 22 cases in 1931-1932 17, or 77.3% were simple, and five, or 22.7% were strangulated. All of the former had surgical repair, with no mortality. The five cases of strangulation present a mortality of eighty per cent, and further comment on them is of interest and value.

TABLE IV

Cases of Strangulated Umbilical Hernia (1931-1932)			5
Operated upon	4	Mortality	80%
No operation	1		
Average age			63 plus
Males 2		Females	3

The patient recorded above as having had no operation was a woman of 67 who had apparently had strangulation for one week, and three days before entrance a superficial rupture of the sac had occurred. She was moribund on admission and died in six hours.

TABLE V

Fatal cases of strangulation	4
Duration of symptoms before admission	
Days	
0-1	2
1-2	0
2-3	0
3-4	0
4 plus	2

The above figures demonstrate the tendency of a large percentage of these patients to procrastinate in seeking surgical aid, and account, in a large degree, for the high mortality. The same fact was observed in our former study, in which we noted that eight out of 12 fatal cases had postponed hospitalization for more than three days.

TABLE VI

Fatal cases of strangulation	4
Length of life, in days after admission	
0-1	2
1-2	0
2-3	0
3-4	1
4 plus	1

TABLE VII

CAUSES OF DEATH

Fatal cases of strangulation	4
General peritonitis	2
Uremia	1
Pulmonary	1

The cases of strangulation during the years 1931-1932 are given below with the greatest brevity.

1. Hospital no. 272942. A female, aged 58, entered with a strangulated hernia 10 cm. in diameter, of 11 hours' duration. Repair under local anesthesia resulted in complete recovery.
2. Hospital no. 318266. A female, aged 67, entered with a ruptured sac of umbilical hernia, about 20 cm. in diameter. The strangulation had existed one week and rupture had occurred three days before admission. She died in six hours, no operation being done.
3. Hospital no. 325347. A male, aged 54, entered with a small hernia, 4 cm. in diameter, strangulated one week. Operation under spinal anesthesia was followed in three days by death from massive pulmonary collapse.
4. Hospital no. 323597. A female, aged 65, entered with a large hernia, 30 cm. in diameter, strangulated for only three hours. Operation revealed general peritonitis, and patient died in 19 hours.
5. Hospital no. 321498. A male, aged 74, entered with a hernia 8 cm. in diameter which had been strangulated 10 hours. He died of uremia 15 days after operation.

The conclusions drawn from the previous paper[5] on this subject were that, of simple umbilical hernias, one in every six will strangulate; of strangulated hernias, approximately one in every three will die; and therefore every patient with simple hernia has one chance in 18 of dying of it. Furthermore, the mortality from operation for simple umbilical hernia is less than three per cent, or not more than one case in 33. Our recent figures bear out and emphasize these findings.

We therefore assert that palliative treatment of simple umbilical hernia with belts and appliances, constitutes a dangerous form of procrastination, and surgical operation is far safer.

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Discussion

CHAIRMAN RICE: Dr. Miller's paper is now open for discussion. Dr. Lanman of Boston will speak.

DR. THOMAS H. LANMAN, Boston: *Mr. Chairman and Gentlemen*—I have been asked to speak on the aspects of umbilical hernias as we see them at the Children's Hospital.

As regards its incidence,—in the last five years we have had about 7,500 admissions to the surgical wards. Of these admissions there have been nearly one thousand cases of inguinal hernia. Inguinal hernia is very common, and in practically all cases we believe operation is needed for cure. During this five-year period there have been admitted to the surgical wards only about fifty patients with umbilical hernia. This, however, does not represent the true comparative incidence of umbilical hernia, for in the Outpatient Department during this period there were 430 cases of umbilical hernia referred to us. Of these 430, only 58 were operated on. That is, about one case in nine, as we see them, need an umbilical repair.

The vast majority, as Dr. Miller has pointed out, can be perfectly satisfactorily cured during infancy by adhesive strapping. In using this method of treatment, there are one or two points that we feel are of importance. The strapping must be continuous. The usual story is that the child has been strapped "ever since it was born", but on close questioning, one finds that the adhesive strapping is put on, is left on, until it falls off, and only then is replaced.

The strap should be one and a half to two inches wide, depending on the size of the child, and should extend well out on either flank. We believe that no button, penny or other pad should be placed beneath the adhesive. The purpose of the strapping is to obliterate the defect by the close approximation of the recti muscles.

In changing the adhesive, one must have the fresh strap ready and not allow the child to strain while it is being changed. In hot weather it is important to change it rather frequently, as the child's skin may become irritated.

When do these cases become operative? Usually, if the hernia is going to close by strappings, it will be closed by the end of a year or a year and a half. Often by that time the hernia ring though not closed is so small that it will scarcely admit the tip of the little finger. These cases we think probably do not need operation. You generally have made as much progress by adhesive strapping as you are going to by the end of a year, especially, if at this time you feel a rather thickened cartilaginous edge of the ring itself. We tend to be conservative with umbilical hernia, and radical with inguinal. That is, if by the second year, the ring is so small that the tip of the index finger cannot enter, we feel that these cases need neither operation nor further strapping. If these cases caused trouble in later years of childhood, we

believe we would know about it, but we do not see them return for further treatment.

The Mayo operation such as is used in the adult, is the one best suited for repair. We have never since 1919 had a case where there has been strangulation of an umbilical hernia in a child, or one in which there have been adhesions of the contents of the sac and the sac itself. In doing the Mayo operation the only modification we use is to make a curved incision below the umbilicus, turning the umbilicus up so as to preserve it. This is especially important in boys.

The larger type of umbilical hernia that we see, we classify as an omphalocele. Here the abdominal contents herniate through the umbilical ring, into the cord itself. They vary a great deal in degree. In the extreme cases almost all the intestines, liver and other organs are outside the abdominal cavity and in the omphalocele. The reason these cases are so difficult to cure is that the abdominal cavity itself has not developed. One has the greatest difficulty in returning the contents of the sac to the abdominal cavity, because of lack of space. In cases where the liver is out in the sac, the prognosis is almost hopeless, although I have had two cases in the last five years that survived in spite of the fact that a large portion of the liver was out in the sac. These cases also, are very likely to have adhesions of the contents of the sac to the sac itself. These cases are emergency cases. The prognosis is very bad especially if the liver is in the sac.

In closing, let me say that though small umbilical hernias are very common most of them can be cured without operation. There are many cases in which the hernia ring may still be palpable after a long period of strapping. If this ring is small I doubt if operation is needed. Certainly we see no evidence that these small rings give rise to any trouble in later childhood.

CHAIRMAN RICE: Dr. Miller's paper is now open for general discussion. Has Dr. Alton of Worcester come in? (No response.) If there is no further discussion, Dr. Miller, would you like to say anything?

DR. RICHARD H. MILLER: I think that we do not need to feel backward about operating on most of these umbilical hernias in adults if we give sufficient thought to the preparation and to the anesthetic. As I have become older I have found that I can do so much more under local anesthesia that I am becoming more and more dependent on it. If I have, for instance, a stout patient and feel some hesitation about the use of ether, I first consider spinal anesthesia and then should I not consider this wise, I would go ahead with local anesthesia. I believe that even a very large hernia can be successfully repaired in this way without undue discomfort to the patient and with a minimum possibility of postoperative complications.

CHAIRMAN RICE: The Chair would like to take advantage of this pause in the program to hear the report of the Nominating Committee, if it is ready. Dr. Truesdale. Apparently the Nominating Committee is not ready to report.

The next paper is The Relation of Trauma to Hernia, by one who has given much thought to this much mooted question. Dr. John J. Moorhead.

RELATION OF TRAUMA TO HERNIA*

BY JOHN J. MOORHEAD, M.D.‡

IN February, 1932, the writer read a paper entitled "Traumatic Inguinal Hernia" at the Mid-South Post-Graduate Assembly in Memphis, and this was subsequently published†.

Since then a number of inquiries have been received bearing upon the ideas then advanced, and today opportunity is presented to amplify and perhaps clarify some points in dispute.

Let it be understood that the topic under discussion is inguinal hernia in male adults and the relation trauma bears to the production of this lesion. There is, of course, nothing new in this inquiry, for the relationship of trauma to hernia has long been a vexing and perplexing problem and there is a vast literature on the subject, medico-surgical as well as medico-legal.

The problem is twofold; the first is an answer to the question "Can Hernia be Caused by Trauma?"; the second question is "If Hernia is not caused by Trauma, can it be aggravated thereby?" In other words we seek to determine (I) *Causation*, and (II) *Aggravation*. Viewed thus our inquiry becomes better defined and the segregation thus apportioned is more likely to result in accord and not discord.

I. *Causation.* Can one act of trauma become the sole producing factor in the development of a hernia? This question is scarcely controversial now, because there is general agreement that no single, isolated or individual act of trauma is causative in a normal individual unless there is actual laceration of the overlying structures. This virtually implies incisional hernia, compound or open wound hernia if you

*Read before the Section of Surgery of the Massachusetts Medical Society at its Annual Meeting, June 6, 1933.

†J. A. M. A. 98: 1785 (May 21) 1932.

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prefer, and that sequence of injury is so rare as to be negligible.

It is universally conceded that a hernia does not and cannot develop in the absence of a preformed sac, and to that degree a hernia is born and not made.

In other words, there is a congenital predisposition or an acquired anatomical defect leading to the formation of the peritoneal pouch, this diverticulum of peritoneum, that we call the sac. In the absence of this peritoneal irregularity, this corrugation of the visceral covering, there can be no protrusion of it through potential openings, and hence there can be no herniation. This doctrine of preformed sac is established by abundant anatomical and surgical proof, and there is hence no controversy in answering question I by stating that trauma is unrelated to the causation of hernia in the absence of demonstrable laceration of the overlying structures.

No amount of cross-examination can shake this fundamental assertion which is founded on the statements of experience of the sages in our profession.

II. Can Trauma Aggravate, Accelerate, Accentuate, Increase or Modify Hernia? The answer is "yes", if the attending circumstances so indicate.

And what are these "attending circumstances"?

There will be no controverting the assertion that the first essential is increase of intra-abdominal pressure. Only one other element could be equally productive, and that would be deliberate or purposeful dilation of the external ring, the inguinal canal or the internal ring.

That very procedure is still accomplished by professionals in certain countries where military service is obligatory and where the presence of hernia is sufficient cause for rejection.

But that contingency does not interest us and it is only mentioned to show how difficult and painful it is to interfere with these well-guarded openings by attempting suddenly to dilate them digitally or instrumentally from below. In passing, let it be stated that these professional dilators often make three attempts before they are successful, and in the process inflict pain enough to make the victim faint. The suggestion and implication is that if deliberate dilation is so painful and disabling in a prepared and desirous subject, how much more painful must be the process in an unprepared subject; in other words, sudden onset by unexpected force must in accredited cases be accompanied by pain.

If intra-abdominal pressure is the prerequisite, we can postulate our problem by the statement that this pressure must be adequate in degree and adequate in effect.

There must be sufficient force to register in terms of sudden expansion of intra-abdominal pressure to produce dilatation of a ring or to produce a protrusion between the rings. There can be no hernia unless there is dilatation of a ring, of the canal, or of muscle and fascial coverings.

Elsewhere* I have called attention to the resemblance of the birth canal to the inguinal canal; the internal os resembling the internal ring, the external os the external ring, the cervical canal resembling the inguinal canal.

If impregnation and growth occur, then intra-abdominal pressure in the one case produces the fetus, and in the other the hernia. But the prerequisite in each case is impregnation, growth, and intra-abdominal pressure.

An obstetrical analogy of this sort is quite apt, and no one familiar with the subject would say that the last pain produced the fetus, although the "last pain" doctrine is often regarded as the producer of the hernia.

Then if intra-abdominal pressure is the essential, what constitutes force enough to be regarded as solely causative? This can be summated in terms of *adequacy* as to site, and *adequacy* of effect. A light blow on the upper abdomen should be less productive than a heavy blow on the lower abdomen. A distant blow, a wrench, a sudden strain, a violent cough, a valiant sneeze, straining at stool, extreme vomiting, lifting, pushing up or pushing against, all of these singly or together have been accused as causative. The relationship of any or all of them has been so apparent so often that practically all of the laity and many of our profession believe that the cause and effect are indisputable.

Let us pursue this inquiry farther. It is certain that many individuals are so built that sooner or later a hernia will develop. This we know because of laxity of the abdominal wall, paucity of muscular and fascial supports, clefts or apertures in muscle or fascia, and a variety of congenital or acquired defects which weaken this section of their anatomy. Thus regarded, hernia is a ptosis, and in reality that is just what it is in many cases. Further, we know that many persons have hernia and know nothing about it. As proof of this the Surgeon General's office informed me that of 2,754,000 men examined for war duty, four per cent of them between the ages of 21 and 30 had hernias well enough developed to cause rejection for military service. If that is so in men less than 30, how many more must there be in the decades beyond 30? Further, one examination may not be enough, for all of us know that the sac may at times be unoccupied by intestine or omentum and thus escape detection, and yet at another time become perfectly apparent. Many times I have had cases referred by a competent examiner who diagnosed hernia, and yet despite all sorts of straining, jumping, pushing, coughing, it has been impossible for me to demonstrate the protrusion stated to have been palpable or visible,

*Moorhead, J. J.: Traumatic Surgery. Ed. 2. Philadelphia, W. B. Saunders Co., 1917 and 1921.

or both. The corollary of this is that the sac may be there but, unless filled, it escapes detection. To be effective as a producing factor, the intra-abdominal pressure must be adequate enough to fill the sac and perhaps propel it enough to bring it into view or into palpatory reach.

To carry out the obstetrical analogy, it must become a dilating agency sufficient to make apparent the presenting part. Now if this element of adequacy prevails, it needs no argument to sustain the view that the effects of this pressure must be adequate also. Now in trauma there is no incubation period; the cause is succeeded immediately by the effect. The sudden onset produces the sudden effect, and there is no interval waiting period in any form of trauma.

What are these effects? Pain obviously is the first and this should register enough and be disabling enough to make the possessor aware of it at once. Even under local anesthesia in a patient dulled by morphine or barbital, we cannot at operation handle the sac or its contents without producing pain. If we cannot do it then, how is it possible for a hernial sac to appear of recognizable size without pain? And if the pain is not disabling, transient, unaccompanied by nausea, imminent syncope, testicular dolor and other appropriate signs, are or are we not justified in saying that the traumatic act in question was incidental and not productive? The answer is self-evident unless we are willing to scrap all we thought we knew surgically and anatomically of this section of the body. Further, if a man comes to me the day after he says he received his hernia by a lifting strain and tells me he had some pain but not much and he kept on working and that night he "found a lump in the groin", can I as an examining surgeon expect to find clinical proofs to link the "strain" with the "lump"? Yes. What are the proofs? Swelling is one, perhaps ecchymosis. Local tenderness is another. Inability painlessly to palpate the protrusion or to introduce the finger into the ring are other signs, as we all know so richly endowed a section will not fail to resent our examination in terms of pain and tenderness. What am I seeking to determine? Actually my patient must have sustained an acute peritonitis if this asserted "Strain" bulged this preëxisting sac to the extent claimed. Now no medical man within hearing will assert that a segment of peritoneum can be violently thrust from its normal habitat without resenting it unmistakably and asserting it by appropriate signs and symptoms.

Let us go on and operate on this patient, note the findings, make thus a biopsy and send the sac to the laboratory.

The patient is under spinal anesthesia and I proceed to do the modified Bassini operation. Assuredly if the condition is recent, a week old or less, I should find corroborative evidences, such as exudate, organized hemorrhage, traumatized muscle or fascia, and an inflamed sac that has been separated from the contiguous cord structures. Now I have operated on several so-called traumatic herniae as early as a few hours after the alleged onset, but never have I found the slightest evidence of recent pathology. The thick or thin sac was adherent, it had to be dissected, the contents were often shaped or moulded suggesting long occupancy, the base of the sac was wide and easily dilated, often the contents were attached by strong bands, and in every respect aside from the history there was not a single vestige of surgical proof to indicate anything recent. Then the sac went to the laboratory, and shortly the report came back disclosing chronic peritonitis and fibrosis of a grade that would take a year or more of friction and circulatory reëstablishment to accomplish. Now this sequence does not occur occasionally, but it is the persistent rule, the inevitable chain of events, and so I long ago gave up the idea that hernia was connected with injury except under the most unusual circumstances. My assistant, W. D. Ludlum, Jr., has analyzed for me 300 consecutive adult male cases, 232 of these being reported in the article published a year ago. This statistical survey of operative cases shows the following:

In three hundred adult male cases, 234 were unilateral and 66 were bilateral; there were 27 recurrent cases. Ninety-six per cent of the men were between the ages of 20 and 60 years; fifty-five per cent between 20 and 40. The average age was 37.5 years. The greatest proportion, thirty-one per cent, occurred in the fourth decade, from 30 to 40.

The duration was given by fifteen per cent as less than one week; by forty-three per cent, less than one month; by seventy-five per cent, less than one year.

The onset was thought to be sudden in sixty-seven per cent; attributed to a definite strain in sixty per cent. The onset was thought to be gradual by thirty-four per cent.

The hernia was found to be direct in twenty-five per cent, indirect in twenty-three per cent and direct and indirect in thirty-six per cent.

Spinal anesthesia has been used in seventy-four per cent and gas ether in twenty-six per cent. In the last two hundred cases, however, spinal anesthesia has been used in eighty-six per cent.

The Bassini operation was used in forty-seven per cent; the Bassini without a cord transplant in forty per cent.

The recurrent group included twenty-seven cases or nine per cent. The time since prior operation averaged six and one-half years. The average duration of recurrence was twenty and one-half months; the average duration of symptoms, four and one-half months.

Complications included one death (0.3%)

from peritonitis due to bladder injury; pneumonia or other pulmonary complication, 6.3% and suppuration, 3.7%.

In sixty-three per cent of the series a pathological report was made. In fifty-two per cent of these inflammatory changes were noted in the sac; in ten per cent, fibrous and inflammatory changes; in sixteen per cent inflammatory changes alone; in twenty-two per cent no changes.

These same figures pertained whether the patient thought the onset to have been sudden or gradual.

Thus far we have alluded to the anatomical fallacy of believing that hernia could by one act of trauma assert itself, and we have also referred to the biopsy evidences at operation, and stated also the pathologist's findings, and thus we have arrayed the anatomist, the surgeon and the pathologist in agreement.

Are there further proofs to add to this disclaimer? Yes, and these are from the book of Experience, the great teacher.

I do not think we are wrong if we estimate that ten per cent of our male patients in the decades 20-70 have actual hernia, and we actually know, as stated, that four per cent in the 20-30 decade have the condition to an easily recognizable degree. If this is so, then a very considerable proportion of your patients and mine treated for other injuries have actual or demonstrable herniae.

Granting this, the inference is that in fracture of the pelvis, of the ribs, of the femur there should be an occasional instance of aggravation of an already existing hernia.

Have you ever been called upon to treat such an aggravation as a concomitant of injuries that inevitably must have produced extreme intra-abdominal pressure? We have all seen crushing injuries of the abdomen and of the chest with and without intra-abdominal and intra-thoracic damage, but I am unaware of any case reports indicating aggravation of hernia. To me this is one of the very real proofs that aggravation even under extreme violence is not a very active factor; and if violence great enough to rupture viscera and fracture a pelvis or hip does not cause aggravation, it is not surgically logical to ascribe aggravation to lesser causes. Let me cite a recent case of a slightly built 36 year old man who fell 30 feet into an elevator shaft, landing astride an iron beam. He sustained a complete external dislocation of his left knee without fracture; he had dissecting wounds of each groin, one of these eight inches long with extensive laceration of muscles; he also had a circular laceration of the penile skin. Now here we might reasonably expect a hernia or some symptoms referable thereto, but there was no such complication, and I have never had a patient who required attention for hernia in the presence of injury contiguous to the inguinal regions.

Another point seems worthy of note and that is the infrequency of strangulation of hernia under the influence of strain or injury. The majority of my cases of strangulated hernia have occurred at night or outside working hours, and incidentally strangulated hernia seems less common than hitherto. If aggravation due to violence was an important factor, then assuredly strangulation as a sequence should be more common.

However, we are accosted by the clinical fact that pain does occur after some definite act or incident associated with intra-abdominal pressure, and that in some cases a lump appears within a few hours or a few days. What causes the pain and what causes the lump? Pain can be due to tugging or pressure on the sac, to gaseous distention of the intestine within the sac, to a rotation or twisting of the omentum in the sac, to pulling upon the adherent contents of the sac, to irritation by pressure upon the iliohypogastric or ilioinguinal nerve supply, to muscular strain, and perhaps also to torsion of the cord.

The lump appears because the sac is occupied by intestine or omentum or both and it then acts like a Barnes' bag to dilate the orifice or the canal which is virtually a prolapse. But as stated, neither the pain nor the lump denotes recent origin unless corroborated by local tenderness, edema and perhaps ecchymosis.

Hernia is not actually an acute surgical entity but in reality a progressive disease characterized by periods of remission and accession. Inevitably the lump will appear if the already mentioned factors exist, and sooner or later peristalsis will balloon the sac enough to make of it a dilating agency. The accused intra-abdominal strain or injury may be the ascribed factor in the opinion of the patient, but the ascribable factor is the normal progression of a chronic disease manifesting itself by an exacerbation.

As physicians we often have to differentiate the ascribed and the ascribable cause. A patient may ascribe his attack of abdominal cramps to a Cape Cod fish dinner (very unjustly I admit), but any Massachusetts physician would assert that the symptoms were ascribable to an exacerbation of gall stones or appendicitis. Assertion is not proof; the ascribed is not always the ascribable in any intra-abdominal lesion, hernia not excepted.

In differentiating then an old and a new hernia there are certain criteria, certain proofs based on professional knowledge to which we should give heed, and of these I would mention the following:

	New Hernia (Acute)	Old Hernia (Chronic)
Type of Injury	Intra-abdominal pressure from contiguous impact	*Ditto* from remote impact
Type of Person	Muscular	Non-muscular; ptosis build; fat
Time of Onset	Immediate	Late
Type of Symptoms	Pain—nausea—tenderness—edema	Slight if any
Examination Findings	Tenderness—swelling—ecchymosis	Absent or slight
Operative Findings	Hemorrhage—edema—no adhesions —no diastasis, slits or lipoma	Adhesions extra- and intra-saccular diastasis, slits and lipoma
Pathological Findings	Acute peritonitis—hemorrhage.	Chronic peritonitis, fibrosis

Now all this has a medico-legal bearing, and we all are beset with doubt in attempting properly to allot responsibility. The Compensation Laws in some states require definite surgical proof before making an award, and the seven criteria named above are some of the main factors in the determination. In most industries a preëmployment physical examination is required and then the applicant is rated as to hernia in some such terms as

1st degree hernia—external ring admits tip of index finger
2nd degree hernia—ditto, plus impulse plus gaping ring
3rd degree hernia—ditto, plus lump
4th degree hernia—ditto, plus lump below external ring.

The question of repeated violence, successive intra-abdominal pressure and continuous or intermittent strain are of course important elements in those predisposed, and in this group it is only a question of time until the process assumes recognizable proportion.

It is, however, surprising to note the frequency of obvious hernia without the possessor being aware of it; but once attention is called to the finding the inevitable comment is "I must have strained myself"; or "I remember lifting something some time ago."

Repeated intra-abdominal pressure is an indisputable causative factor, and incidentally under spinal anesthesia with the canal opened, it is interesting and instructive to ask the patient to cough and in so doing the sac is ballooned just as it is if the patient becomes nauseated during the operation.

My conception of the development of hernia is that the preformed sac enlarges, acts as a dilating wedge and at the appointed time presents itself as a completed product palpable and perhaps visible to the physician and to the patient. That it may occur as the outcome of a single traumatic agency is not to be decided until the extent of the intra-abdominal increase is estimated in terms of *adequacy;* if adequate, then the ensuing symptoms are immediate and demonstrable.

In passing it is pertinent to say that the surgical cure of hernia has also a bearing on its causation and no experienced operating surgeon has failed to note the extensive pathology involved, the nature and diversity of which required years and not days or weeks to produce. Further, there is often congenital absence of normal protective barriers such as a wide inguinal triangle or a very thin or imperfect conjoined tendon. Again, other cases show wide separation in the fibres of the internal oblique, and definite slits or thinning of the external oblique fascia. Not infrequently varicosities of the cord act as the dilating agency, to say nothing of that large group in which lipoma of the cord may act as a wedge to force apart the contiguous structures. Obviously none of these findings are in consonance with any recent occurrence, for all of them are congenital or slowly acquired.

It has been said that direct hernia is more likely to be acute then indirect hernia, and that scrotal hernia is the patriarch, the ancient of ancients. Bilateral hernia assuredly is practically never acute, and about 60 per cent of my patients have hernia on both sides. Further, a direct and indirect hernia on the same side scarcely can be called acute, for to produce such a sequence by one act of violence implies great force and inevitably great effect, in terms of disabling pain that should send the patient into a state of shock.

It is a surgical fallacy to believe or assert that any single act of trauma can without symptoms bulge the sac and propel it through normal openings or along the canal in an area so richly endowed with a vascular or neural supply, and so well buttressed by strong muscles and fascia. The sac is a diverticulum, and when it becomes suddenly bulged it may be regarded as producing an acute diverticulitis, and if so it registers like any other diverticulitis, namely, by nausea, pain, and tenderness. The appendix is the best known diverticulum and the cardinal symptoms of appendicitis are the well-known trinity of nausea, pain, and tenderness, just as in "hernia-itis". I am aware that "traumatic appendicitis" is also an interested party in medico-legal controversies, but I have never seen it at the bedside, nor heard it discussed in reputed surgical societies by men whose opinions you and I value. It should have about as much scientific standing as "traumatic tonsillitis" due to hanging or a blow on the neck.

It is opportune for medical societies such as this to go on record as to their belief in respect to this age-old bugaboo and to definitely state

what is and what is not a so-called "Traumatic Hernia".

SUMMATION

1. Hernia is never *caused* by injury; the preformed sac is always an antecedent.
2. Hernia can be *aggravated* by injury if the source and symptoms are *adequate*.
3. Immediate disabling pain is the chief symptom and this is associated with nausea, tenderness, swelling and other manifestations.
4. At operation the findings usually denote an ancient process as indicated by adhesions, extra-saccular and intra-saccular.
5. The pathological examination of the sac demonstrates chronic peritonitis and fibrosis.
6. Hernia is a chronic progressive disease, a ptosis, a diverticulum, and not an acute surgical entity except in very rare instances.
7. A large proportion of our adult male population have herniae and do not know it; and we as surgeons do not know it either when we are treating them for contiguous injury grave enough to cause aggravation if trauma is regarded as a common aggravating factor.
8. Like osteomyelitis, once a hernia always a hernia, and it is subject to periods of accession and remission.
9. If trauma is to be accused and found guilty, then there is always plenty of reliable evidence, and this need never be medico-legally seductive or speculative, but rather medico-surgically deductive and positive.
10. Trauma has burdens enough without being saddled with this ancient heritage which modern traumatology has cast into the discard along with grape seeds as a cause of appendicitis.

DISCUSSION

CHAIRMAN RICE: Dr. Daniel F. Jones of Boston will open the discussion.

DR. DANIEL FISKE JONES, Boston: *Mr. Chairman, Gentlemen*—It is difficult to discuss a paper when one agrees with all that has been said by the speaker.

I have for years seen cases that were said to be of traumatic origin, but no patient has ever given any history of a sudden attack of pain, nor has he ever spoken of unusual tenderness following the injury. Rarely is anything said by the patient until some weeks or months afterward when a bunch is discovered by the patient or by the physician.

I believe it is possible for a hernia to appear suddenly at the time of an injury when there is a congenital sac which has never closed entirely at the internal ring, but I believe that in these cases there is always definite pain at the time when the internal opening is suddenly dilated. Stretching peritoneum always causes pain.

Dr. Irving Clark in an article about one year ago spoke of the examination of the men in one of his plants. He divided the hernias, and those with a large external ring and an impulse against the finger when put into the canal, into several degrees of hernia. It is of interest to note that among 289 men with what he called a first degree hernia, six and three-fourths per cent developed what he considered a second degree hernia in from one to ten years, while Dr. Moorhead has just stated that ten per cent of people in general have hernia. This heavy work would not, therefore, seem to have any great effect upon the development of hernia. I would much prefer to operate upon a man who does heavy work continually than upon one who takes no exercise at all.

It has been my teaching that the size of the external ring has nothing to do with the development of a hernia, and very few can determine as to whether there is a beginning hernia by putting the finger into the canal through the external ring. It is almost impossible to decide whether it is the posterior wall of the canal or a sac striking the finger.

The anatomy, and particularly that of the posterior wall of the canal, of the individual has much more to do in determining as to whether there is to be a hernia than acute trauma or the continual trauma of heavy work.

Race, because of anatomical variations in the various races, has much to do with the formation of hernia. A large percentage of Armenians have a bulging posterior wall of the canal due to its weakness and therefore are liable to hernia. Dr. Clark noted that a large percentage of the cases among his workers were in Italians.

I agree entirely with Dr. Moorhead that there is no such thing as a real traumatic hernia, unless there is on very rare occasions a congenital sac which has not quite closed at the internal ring and through which a piece of bowel might be forced. In such a case there must be pain.

CHAIRMAN RICE: Dr. Sweet of Springfield, who was to continue this discussion, is unable to be present. Dr. Moorhead's paper is now open for general discussion. Dr. Lund, you must have something to say.

DR. FRED B. LUND: I fully agree with all that Dr. Moorhead and Dr. Jones have said. I have never seen what I could call a traumatic hernia where there was evidence of ecchymosis and edema and pain immediately after the accident which is supposed to have caused the hernia. They are practically always congenital and always due to the peritonal pouch.

CHAIRMAN RICE: Does anyone else wish to discuss this paper? Would you like to close this, Dr. Moorhead.

DR. MOORHEAD: I was very much interested in what Dr. Jones had to say in regard to the working class. That has been so much impressed

on me that recently I have been allowing my patients to get out of bed following operation on the seventh or eighth day instead of keeping them in bed ten or twelve days. I have learned that in joint injuries, immobilization instead of mobilization seemed to produce atrophy or inertia, and hence after herniotomy to remain inert in bed for twelve or fourteen days, we ran the chance of having atrophy from disuse, and a sudden strain was then very likely to produce a recurrence, whereas it would not be so likely to recur if the patient were permitted to get up a day or two earlier.

CHAIRMAN RICE: The next paper, Recurrent Herniae, is a problem that strains veracity and taxes ingenuity. Dr. Marble.

RECURRENT HERNIAE*

BY HENRY C. MARBLE, M.D.†

WHEN I started to prepare this paper, it seemed to me that "Recurrent Herniae" was rather a shopworn subject. Much had been written without progress, but as I look over my own cases and study the literature and analyze my own results, it seems to me that there is another method of approach which might be helpful in reducing the number of so-called recurrences following inguinal and femoral herniae.

The surgical literature is filled with articles and reports by competent surgeons in the matter of herniae. The surgeons are experienced and the surgery is well done, but in all the reports there are recurrences varying from 1.5 per cent up.

TREATMENT OF RECURRENT HERNIA—HOOK U. S. N. MED. BUL. 1931)

Total primary inguinal		639	
Recurrent inguinal		76	
Femoral		10	
Indirect hernia	360—56%	12 recurrent	16%.
Direct	191—30%	58 recurrent	76%.
Saddlebag	88—14%	6 recurrent	8%.
Bilateral	160—33%	14 recurrent	23%.

Recurrence Rate of other Men
Coley—1918—5.7% 1924—8.7%.
Taylor—1920—Indirect 5.6% Direct 19.08%.
Erdman—7.5% total.
Lyle—10.3% catgut—5% fascia.

Brooklyn Naval Hosp.—10.6% of all herniae admitted were recurrent. Sixty-five per cent recur in first 6 mos., 80% within the first year, 6½% after 2 years.

Causes of recurrence.
1. Wound infection.
2. Failure to get a direct sac in an indirect hernia.
3. Approximation of muscles and fascia under tension.
4. Failure to unite the internal oblique to Poupart's low down.
5. Leaving fat along cord.
6. Injury to inguinal nerves.
7. Leaving internal ring too large.
8. Postoperative vomiting and coughing.
9. Returning to labor too soon.
10. Tying knots too tight?

*Read before the Section of Surgery of the Massachusetts Medical Society at its Annual Meeting, June 6, 1933.

†Marble—Surgeon to Out Patients, Massachusetts General Hospital. For record and address of author see "This Week's Issue," page 607.

Hook does not believe in transplanting the stump and gives a case where this procedure resulted in tearing off of tie with hemorrhage.

TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION, GIBSON AND FELTER, 1930

Number of herniae—1878
Number of recurrences—48

Ages	No. of Cases	Recurrences	% Recurred
1-10	153	1	.6
10-20	193	0	0
20-30	467	13	2.7
30-40	374	15	4.0
40-50	276	13	4.7
50-60	128	4	3
60-70	22	2	9
70-80	5	0	0

Direct Hernia	427	28 recurred	6.5%.
Indirect Hernia	1451	20 recurred	1.3%.
Total single hernia	1618	25 recurred	1.5%.
Total bilateral hernia	260	23 recurred	8.9%

(Bilateral hernia done at one operation has higher % of recurrence.)

72.9% recur in first nine months.
27.1% recur over nine months.

Greatest cause of failure to get the complete sac especially in direct herniae, and failure to recognize a direct sac along with an indirect hernia.

INGUINAL AND FEMORAL HERNIA FOSS AND HICKEN, AMERICAN JOURNAL SURGERY, 1932

Total patients—778.
Unilateral inguinal—558—5.9% were direct.
Bilateral—160 (22%).
128—Bilat. indirect inguinal.
17 Bilat. direct.
15 Saddlebag.
8 were sliding (Sliding more common in bilateral hernia.)

Femoral—60 (7.7%) were women.
13 had had strangulation.
20 were admitted with strangulation.
2 were bilateral.
10% had been previously operated for an inguinal hernia. Femoral hernia not recognized. There were no recurrences.

Inguinal recurrence rate—1.6%.
73% within first 6 months.
Treatment—Bassini operation best method
Failure. Recurrence due to faulty union between Poupart's and internal oblique. They use *Black Silk* in the suture.
Anesthetic—Spinal by far the best.
Do not like Gallie method.

MY OWN EXPERIENCE

Cases	234	
Single hernia	193	
Double herniae	28	
Femoral herniae	13	
Recurrent	5	(On admission)
Strangulated	8	
My own recurrences	9	
Deaths	1	(Pulmonary Embolus thirteenth day)

FREQUENCY

As a great majority of my cases are industrial cases, I looked in the report of the Massachusetts Industrial Accident Board of 1931 and found the following figures.

"For the year ending June, 1931 throughout the State of Massachusetts 1,335 herniae cases were reported. Of these 1,335 cases five were reported fatal and two permanently disabled."

I have estimated the cost of these cases to be about $250 per patient. The total cost to this State would be in the neighborhood of $350,000 for herniae alone.

CAUSES OF MY OWN RECURRENCES

1. The Slipping of the Transfixion Stitch at the Neck

Many times when operating upon a recurrent hernia, I have found a complete sac of an indirect hernia present. The records of the cases stated that there had been a previous operation for an indirect hernia.

Case Report (Case Z): In 1926 I operated upon a patient for a large indirect inguinal hernia. The sac was isolated and carefully dissected. The neck of the sac was closed by a chromic catgut transfixion stitch and the sac was entirely removed. Recurrence in about nine months. At the second operation upon reopening the cord, I found a sac almost as large as the former and much more adherent to the surrounding tissues. At the neck of the new sac I was able to demonstrate the site of my former transfixion tie. It was evident that the tie had untied itself and come off probably before the patient had left the Hospital following the first operation. The omentum had passed through the opening and down the cord and a new serous sac had formed itself about the protrusion of the omentum.

Since that time I have discontinued the practice of transfixing the neck of the hernia sac. After the sac is removed and the neck tied, I then suture the edges of the peritoneum completely and carefully together as one closes the peritoneum elsewhere with a continuous stitch.

Case T: This was an indirect inguinal hernia. His recurrence was an indirect hernia. I believe this was due to the failure to completely excise the hernia sac plus perhaps the failure of the stitches about the neck of the sac to hold.

2. Direct Hernia in Addition to Indirect Hernia

This type of hernia is sometimes called a saddlebag hernia. The indirect hernia is operated upon. The indirect sac is completely removed and the sac closed. The abdominal wall is repaired with all the structures over the cord bringing the cord out at the bottom of the inguinal canal. This type of repair is fairly common. Later there is a recurrence of the direct type, the protrusion coming out through Hesselbach's triangle and through the newly formed internal and external rings. Whether there was a direct hernia present at the time of operation or whether the direct hernia recurrence was a result of the type of operation done, I do not know, but it is in my experience the commonest type of recurrence. Such a type of recurrence does not so frequently follow the Bassini type of repair and when it does occur it comes out between the stitches of the internal oblique muscle to Poupart's ligament and is usually at the bottom of the wound.

Case W (Slide shown): This is a direct after an indirect hernia. The patient was operated upon for an indirect hernia. He was repaired after the Ferguson method. He then had a distinct large direct hernia, this time he was repaired by the Bassini method using fascia lata.

(A slide was also shown of an indirect inguinal hernia.)

Case H: This was a recurrent right inguinal hernia. This patient had a large direct hernia on the left and a small indirect inguinal hernia on the right. He was operated upon by the Ferguson method on both sides in July, 1927. He then had a direct recurrence following the indirect hernia. This is the only recurrence in double hernia in this series.

3. Large Herniae in Fat Elderly People

This is the type of hernia which is particularly difficult to handle. When this hernia is operated upon and a large indirect sac resected, one has only soft weak tissues to utilize in repairing the abdominal wall. I believe in all of these cases permission to remove the testicle or transect the cord should be obtained before the operation, that this procedure should be used more frequently and that at the time of original operation the repair should be reinforced with fascia lata.

I have four such recurrences two of whom were reoperated upon and had the testicle removed and have remained well. The other two have never been reoperated, but when they are this is the procedure which will have to be followed.

Case A: This patient was operated upon April 14, 1924 for a left indirect inguinal hernia. He was a large fat man. He then had a recurrent left indirect inguinal hernia and was again operated upon December 5, 1924. At this time the left testicle was removed.

Comment: This was a recurrence of an indirect hernia in a heavy fat man. I believe the

recurrence could have been prevented if the testicle had been taken out or the cord transected at the time of the first operation.

Case K: This is an old man very fat with an enormous incarcerated hernia. The structures were weak and he recurred.

Comment: At the time of the operation this man's testicle should have been removed or the cord transected and fascia should have been used in the repair of this hernia. He has never been operated upon since.

4. Femoral Herniae

In 1925 I saw a patient (Case R) who had been operated upon for an inguinal hernia. He was operated upon a second time. There was recurrence and he was operated upon again, this time by the Gallie method using fascia lata. There was a third recurrence and it was for this recurrence that I saw him.

He was admitted to the Faulkner Hospital and after two days' preparation, under novocaine infiltration anesthesia, was operated upon. The scars were excised and the entire inguinal region exposed. The patient was asked to cough and a small tumor the size of an English walnut presented itself near the symphysis pubis at the bottom of the hernia wound. This tumor could not be palpated when the patient was lying down. The tumor was then dissected out to the amazement of everyone present and it was found that the neck of the sac came through the femoral opening and that it was in fact not an inguinal but a femoral hernia. Since that time I have seen three such cases that have been operated upon for inguinal herniae in which a femoral hernia was found to be present at the time of operation for recurrence.

Case P: This man was operated upon for a direct inguinal hernia under spinal anesthesia. He had no indirect sac but a definite direct inguinal hernia. The hernia was repaired by the Bassini method. The recurrence was found to be a femoral hernia.

Comment: The repair of the hernia pulling up Poupart's ligament may have weakened the femoral opening or the femoral hernia may have been present all of the time and this was a failure of diagnosis.

I want to call your attention to this position of the sac in certain femoral herniae. Instead of presenting itself opposite the femoral canal, the hernia dissects upward and appears opposite the external inguinal ring. When the patient lies down it cannot easily be found unless one has knowledge of its presence, and at operation exposes the entire inguinal area together with the external femoral opening. In addition the patient must be conscious and able to cough at the proper time.

(Tasche-*Arch. Surg.*, Oct., 1932)

FEMORAL HERNIA

Etiology—Chiefly accepted theory is that of traction not from pressure, i.e., intraäbdominal. The large part of femoral hernia is fat and may also have a filament of peritoneum. The fat probably first forms and is either forced along by muscular action, or because of its semi-fluid nature acts as a water-hammer and forces its way down under Poupart's ligament. The diverticulum of peritoneum is thus dragged down and is thus secondary to the fat. As abdominal content enters the sac, the pressure causes atrophy of the outer layer of fat, and thus the nature of the hernial coverings changes.

Incidence—University Hosp. had 130 inguinal herniae to 52 femoral. 21:1.
More common in females. 1.6 in adults, 1.2 in children.
Most common between 25-45. Rare in children.
Most common on right side (60%).
More common in multiparae than in unmarried women.

Contents of Sac
12 cases had empty sacs.
6 contained normal omentum.
6 contained strangulated omentum.
0 contained normal bowel.
4 contained strangulated bowel.
Of these 28 cases 20% were operated for strangulation.
Forty per cent of all strangulated herniae are femoral, but these are only 5% of all herniae, i.e., they are 8 times more apt to strangulate.

Recurrences
In 25 operated cases at University Hospital there were 2 recurrences.
Watson in 1444 cases found recurrence in less than 5% of cases.
Noble says recurrence is from 25-30%.

Method of Repair
Pott by just removing sac got 63.3% cures, while with closure of ring cure was 76.4%.
Bull in 1890 closed 13 sacs, traced 8 cases and had 3 recurrences.
Bresset studied 395 femoral herniae and in 232 cases without closure had 29% recurrences with closure 8.6%. Keynes closed the sac from a laparotomy in 10 cases and had 6 recurrences.

DIAGNOSIS

1. There must be a visible and easily palpable tumor.
2. Pain is not a sufficient symptom to warrant operation.
3. An impulse on coughing is not adequate to justify a diagnosis.

COMMENT

Dr. E. L. Young has pointed out that pain in the groin is not a symptom of hernia.

I have never yet seen a case of so-called iliopsoas bursitis.

PREPARATION FOR OPERATION

1. Patients to be operated upon for herniae should be admitted to the Hospital two days before operation.
2. They should be shaved, given a bath and two soap and water and alcohol scrubs each day.

3. The thigh should be prepared for removal of fascia.

I believe my incident of wound infection has been reduced since beginning this procedure. The area of hernia wound is difficult to clean and such a prolonged preparation as described, I believe, shortens the patient's stay in the hospital because of the reduction of incidence of sepsis.

ANESTHESIA

For several years for hernia operation, I have used local infiltration of novocaine or regional spinal anesthesia. It is especially important, as I have said, that the patient be conscious and coöperative and able to cough at the proper time when the wound is wide open thereby demonstrating the protrusion or protrusions. This is impossible with a general anesthesia.

OPERATION

1. Complete excision of the scar.
2. Have the patient cough and produce the tumor or tumors.
3. Take apart the previous repair and again demonstrate the protrusion.
4. Open and remove the sac or sacs.
5. Repair the recurrence preferably by the Gallie method. In certain cases complete removal of the testicle is indicated and often necessary.

METHOD OF OBTAINING FASCIA

One of the objections to the use of fascia lata in the Gallie method of repair seems to be the difficulty of obtaining the fascia and the necessity of a long incision on the thigh. Several methods have been devised for taking fascia but the instrument devised and demonstrated by Dr. Philip D. Wilson of Boston, seems to me to be the most satisfactory. With this instrument the fascia as long and as wide as necessary may be obtained through a small incision not more than one inch long. It reduces the time of operation and makes the means of obtaining fascia very simple.

SUMMARY

Recurrences of herniae following repair are common, averaging about five per cent. In analyzing nine of my own recurrences I find they are explained as follows:

1. Failure properly and permanently to close the neck of the sac in the indirect hernia.
2. Failure to recognize a saddlebag hernia, that is to repair only the indirect and not the direct hernia.
3. Failure to remove the testicle or transect the cord and use fascia lata at the first operation in fat people with large herniae.
4. Failure to diagnosis a co-existing femoral hernia at the time of operation.

DISCUSSION

CHAIRMAN RICE: Dr. Ralph W. French will open this discussion.

DR. RALPH W. FRENCH, Fall River: Dr. Marble has called forcibly to our attention the apparent need for more careful examination of a hernia before as well as during the operation.

To overlook a second sac of a hernia is comparable with leaving a stone undiscovered during an operation for nephrolithiasis.

At the Truesdale Hospital during the past ten years 503 patients were operated upon for inguinal hernia. Of this number we have succeeded in tracing by a routine "follow-up" letter, by examination, and by telephone, 479 cases or 95 per cent. Ninety-three cases were bilateral, making a total of 572 hernias traced.

In this series of 572, 31 were recurrent. While 17 had been operated upon at other hospitals, 14 were our own recurrences, giving us a percentage of 2.5.

Over this ten-year period there were four deaths, in three of which the hernias were strangulated. The fourth patient died 17 days after operation from coronary occlusion. All four were 70 years of age or over, giving us a mortality rate of .9 per cent.

Eighty-four per cent. of these 572 hernias were operated upon by one surgeon.

In studying these 31 recurrent cases, I have not found any common cause that could be applied to the majority.

Seven hernias recurred directly following an accident. In these the time after operation varied from two months to four years; but most of them were within a year.

Three patients that were operated upon for indirect hernias returned within two months with direct hernias, which, I feel, must have been present and overlooked at the first operation. This observation bears out Dr. Marble's contention.

Three cases presented a recurrence external to the operative scar and halfway between the internal and the external ring. At operation it was found that the edge of Poupart's ligament had apparently been torn away. In about one-third of the recurrences there was a history of sepsis, by which I mean an accumulation of serum in the wound, or frank pus.

The type of operation, I feel, is an important factor in a discussion of recurrent hernias. For example many indirect hernias are undoubtedly cured by removing the sac regardless of the way in which the rest of the repair is effected; but in the direct type of hernia, it is generally agreed that the best results follow transplantation of the cord. In such instances, if the sac

is small and the tissues are strong and well developed, the Bassini operation will usually give a good result.

In a direct hernia, however, this condition of the tissues is seldom present and ordinarily it is advisable to obtain added support, as is done in the Andrews' modification, by suturing the inner edge of the external oblique to Poupart's ligament. In some cases of the direct type we even go a step farther and suture the outer edge of the external oblique under the cord, leaving that structure covered only by the subcutaneous fat and skin.

Therefore, I feel that failure to recognize the fact that no one type of operation is suitable for all types of inguinal hernia is undoubtedly responsible for many recurrences.

CHAIRMAN RICE: Will Dr. Ober of Springfield continue this discussion? Dr. Ober? He seems not to be here. Dr. Marble's paper then is open for general discussion. Dr. Whitaker.

DR. L. R. WHITAKER, Boston: I would like to say a word about the use of silk sutures in the repair of recurrent hernias, in fact of any hernias. It seems to me that the one essential for a cure of a break in the abdominal wall is repairing that break in just as solid a manner as possible. Incidentally I will say that the method of handling the sac is of little consequence. The peritoneum is a very weak distensible structure. A solid repair of the wall is attained with far greater certainty by the use of silk than with catgut. Animal experimentation shows that even chromic catgut may be softened or absorbed in one to two weeks, at the time when support is most needed. Any increase of intra-abdominal pressure at this time may break open the wound. I have had a patient, in whom chromic no. 2 catgut was used, who broke open an abdominal wound by coughing one week after the operation. Not a shred of catgut could be found in the wound and there were spaces of tissue liquefaction half a centimeter in diameter where the stitches had been. You are never secure in repairing a hernia with catgut.

On the other hand, silk holds forever. Introduced years ago by Halsted it has been used by many surgeons with excellent results.

I know that a good many are prejudiced against silk because they think that it is a foreign body in the wound and that an infection may occur which is difficult to handle. With proper care infections in hernias are rare, and if they do occur are more likely to affect the fat than the better blood-supplied structures which you repair. I have had two cases of fat infection recently which healed readily without disturbing the structures holding the silk. If one is fearful, he can use silk for suturing the deep layer (internal oblique to inguinal ligament) and catgut for the superficial layers.

The neurological surgeons have for a long time used silk in the repair of their scalp wounds. Infections are extremely uncommon. These wounds are sometimes subject to a large amount of tension, yet they hold very well.

If this same principle is applied to the repair of hernias—using a large number of silk sutures, with less tension on each one, which stay there forever, a permanent closure will be attained.

I prefer silk to fascial sutures except for the rare case where the opening cannot be closed by approximation of tissues of the part. With numerous permanent silk sutures, considerable more tension can be applied safely than with evanescent catgut. This often makes filling in with fascial strips unnecessary.

It seems to me that the best method for repairing any severe hernia is again that of Halsted, suturing all layers securely under the cord, transplanting it to the subcutaneous tissues. The danger point at the external ring is eliminated. One trick which can be applied to make the opening at the internal ring more secure is to sew the internal oblique to Poupart's ligament up to the cord; then, in repairing the external oblique, carry the suturing laterally over the internal ring so that the cord emerges in an oblique manner upward and outward. This forms a valve-like device. When intra-abdominal pressure increases, the layers are approximated rather than separated. The external oblique should not be sutured lateral to the emergence of the cord on account of danger of pressure on its vessels.

It seems to me that by this method of using a large number of silk sutures, which stay there permanently, fixing all the layers under the cord solidly to Poupart's ligament, you have the best possible chance of preventing recurrence of an inguinal hernia.

CHAIRMAN RICE: Does anybody else wish to discuss Dr. Marble's paper? If not, the last paper, Hernia from the Compensation Insurance Standpoint, by one who sees the other side and pays the bills, Mr. James H. Holland of Boston, district manager of the Liberty Mutual Insurance Company.

HERNIA FROM THE COMPENSATION INSURANCE STANDPOINT*

BY JAMES H. HOLLAND†

"HERNIA from a Compensation Insurance Standpoint" is indeed a subject that has been so widely discussed and written about as to make an ordinary claims man tremble at being called upon to discuss it before a body such as this. I assume, however, that your invitation means that you are interested in the reactions of at least one insurance company representative to the many phases of this highly important subject.

"In my opinion 98% of all cases of hernia cannot be classed under the head of traumatic hernia but are potential herniae and therefore do not come under a Workmen's Compensation Act," says Dr. R. Robinson Duff of Chicago, an eminent medical authority. The same doctor says, "Any intra-abdominal pressure sufficient to cause a tearing or separating of the abdominal wall great enough for a hernia to appear, would give rise to the following symptoms:—history of sudden onset; over lifting or severe strain; shock varying in degree; patient seeks immediate medical aid; pain—patient found in stooping posture, weakness, nausea, and sometimes vomiting; swelling varying in size, marked tenderness over entire area." Pember and Nuzum in an article written some years ago say, "Traumatic hernia is not likely to be produced by a blow which is short of lethal, unless a puncture wound is produced."

I have read hundreds of articles and opinions written by leading doctors and surgeons who have obviously made a thorough study of this perplexing subject, and the vast majority of them are most definite in expression of a positive opinion that a true traumatic hernia is exceedingly rare. With this in mind the cost to industry annually of hernia is appalling. Any man engaged in the handling of compensation insurance in this Commonwealth will promptly state that no other single type of disability is so costly. Many of us dealing with these cases every day have come to feel that all that is necessary for the claimant to prove is that the hernia is actually present, and that the nature of the work engaged in could have been the cause. Whether it actually was or not seems not to matter.

A report of the New England Surgical Society Committee on Traumatic Hernia some years ago has the following excerpt. "We feel that the present day attitude of making someone other than the disabled individual pay for disability for which no one is responsible, has been carried rather further in the case of hernia than with most other alleged injury. The consensus of surgical opinion points very definitely to the conclusion that hernia as a result of a single injury or strain is of extreme rarity. In view of the chaotic state of opinion respecting the etiology of hernia which obtains, not only with the laity but with a considerable proportion of the profession, as well as with courts and compensation boards, it seems wise to lay down certain criteria for determining what constitutes true traumatic hernia. We believe these to be essentially as follows: 1. Immediate descent of the hernia following the alleged cause, be it injury or strain. 2. Severe pain in the hernial region. 3. Marked prostration. 4. Symptoms of such severity that the individual calls attention to his condition within the first 24 hours. 5. No previously existing hernia at present site. 6. Trauma or strain must be adequate. Where two or more of these criteria are lacking the case cannot be justly maintained to be one of traumatic origin. We therefore report that in our opinion this Society should place itself on record as subscribing to the following: 1. True traumatic hernia is extremely rare, that is, most herniae are of very gradual development. 2. An individual presenting a claim for hernia in order to have his claim upheld, must be able to furnish proof of having had at least four of the six criteria above laid down. 3. All other cases may be classed as disease, either of congenital or occupational origin and are not the result of an individual injury or strain." It seems to me that this is a fair, concise, and reasonable position. If followed and adhered to, an unjust and highly expensive charge on industry generally would be eliminated. What do we find as a matter of practice? Case after case goes before the various industrial boards and commissions, doctors testify as to the conditions in the particular case, and based largely on their testimony and opinion, award after award is made for alleged traumatic hernia. Members of commissions and boards, and undoubtedly the judges of the courts are at a loss to know how to properly adjudicate this oppressive problem. The situation in many states of the union is chaotic. There are now to be sure some states in which the members of the workmen's compensation commissions have apparently made a scientific study of the subject before formulating any rules, and in these states the subject is treated in a most fair-minded and judicial manner. In many other states, however, the rulings are apparently based on the old and long discarded ideas as to the etiology of hernia with the result that a great financial loss to in-

*Read at the Annual Meeting of the Massachusetts Medical Society, before the Section of Surgery, at Boston, June 6, 1933.

†Holland—New England District Claims Manager of the Liberty Mutual Insurance Company, Boston. For record and address of author see "This Week's Issue," page 607.

dustry, and in the end a distinct harm to the individual, is brought about.

Recognizing the difficulties and injustices which have arisen, the legislatures of many states have enacted definite sections or amendments to the compensation laws clearly defining the circumstances under which disability for hernia shall be compensated. For example, section 33, 15 B of the Missouri Compensation Law says, "In all claims for compensation for hernia resulting from injury arising out of and in the course of the employment, it must be definitely proved to the satisfaction of the Commission; first, that there was an accident resulting in hernia; second, that the hernia appeared suddenly accompanied by intense pain; third, that the hernia immediately followed the accident; fourth, that the hernia did not exist in any degree prior to the accident resulting in the injury for which compensation is claimed." Kentucky has a section exactly similar to this. The Pennsylvania Law as amended says, "Hernia shall be considered as a physical weakness or ailment which ordinarily develops gradually and shall not be compensable unless conclusive proof is offered that the hernia was immediately precipitated by such sudden effort or severe strain that; first, the descent of the hernia immediately followed the cause; second, that there was actual pain in the hernial region; third, the above manifestations were of such severity that the same were immediately noticed by the claimant and communicated to the employer or a representative of the employer within 48 hours after the occurrence of the accident." The New Jersey Law says, "Inguinal hernia is a disease which ordinarily develops gradually, being very rarely the result of an accident. Where there is a real traumatic hernia resulting from the application of force directly to the abdominal walls, either puncturing or tearing the walls, compensation will be allowed. All other cases will be considered as either congenital or of slow development and not compensable, being a disease rather than an accidental injury, unless conclusive proof is offered that the hernia was immediately caused by such sudden effort or severe strain that; first, the descent of the hernia immediately followed the cause; second, that there was severe pain in the hernial region; third, that there was such prostration that the employee was compelled to cease work immediately; fourth, that the above facts were of such severity that the same were noted by the claimant and communicated to the employer within 24 hours after the occurrence of the hernia."

These specific provisions of the laws of the states referred to, show conclusively that, with industry and the medical profession actively coöperating to make clear-cut and defined the real issue involved in this all-important subject, justice and equity can and will prevail. The elementary principle of workmen's compensation is to place on industry the responsibility of caring for disability brought about by the breaking down of the physical structure in any manner due to the operations of industry itself. I do not feel that it was the intention of the fathers of such legislation to make of the laws a means of charging industry with all the ailments of mankind. I likewise am firmly of the belief that in no other field has the purpose of compensation been carried farther afield than in the handling of hernia.

The Nevada Compensation Commission rule says, "Medical Science teaches now what it has taught for the past twenty years and now accepted as a medical and scientific truth, corroborated as such by the foremost surgeons and anatomists of the world, that is, that hernia or so-called rupture is a disease ordinarily developing gradually and is rarely the result of accident." Dr. Lauffer writing in August of 1919, at Pittsburgh, Pennsylvania, in the Magazine known as *Industrial Hygiene* states with reference to the Pennsylvania situation existing at that time relative to four thousand herniae having been compensated for in the first three years of the compensation law that, "This number of cases represents more than the real cases of traumatic hernia that have occurred on the western hemisphere since Columbus."

The cost of workmen's compensation insurance has increased steadily in our own Commonwealth, due largely if not entirely to the liberalization of the Act itself by legislative amendments and board and court decisions. The Supreme Judicial Court has said that the Act is to be liberally construed and from the outset it has so done. F. Robertson Jones in an excellent address entitled "Ominous Abuses Threatening the Insurability of Workmen's Compensation" said, "Widely and rapidly liberalizing changes in the compensation law system will, if not properly and effectively abated, result in my opinion, in creating under these laws a situation of uninsurability in so far as insurance companies are concerned; an unbearable burden upon industry; and ultimately an intolerable situation in the case of the general public. Unless some drastic action is taken soon to bring the workmen's compensation laws of this country back to a status of reasonableness and to correct the many abuses incident to their administration and enforcement, I am fearful that there will be a complete breakdown of the whole system so laboriously developed during a period of over twenty years. The system itself is one of the most socially beneficent plans for the relief of human suffering which has ever been devised. It is, however, a system which is law operated and law controlled and therefore suffers from the dis-

abilities which are so common in efforts to establish by law, rules and regulations for the distribution of money benefits among the members of a particular class. The tendency in all such cases is for greater benefits and consequently more money; and this tendency is accentuated by over-zealousness on the part of friends, by the efforts of professional politicians to exploit the system, and by the resultant unwarranted extension of its provisions to situations to which it was not designed or prepared to apply. In my opinion industry cannot long stand the increasingly heavy burden on it by the liberalization and enlargement of the coverage and the benefits of the compensation system as it now exists in most of the states. On the other hand, insurance companies cannot long stand the drain on their resources caused by the failure of premium rates to keep pace with losses, and the inability to charge and collect sufficient sums to pay the ever-increasing benefits, awards, and necessary expenses."

What then is the remedy? In states such as Massachusetts where to date no specific provisions have been incorporated in our law I believe it is the duty of industry, the medical profession, and the insurance companies to spread broadcast a clearer knowledge of the well-known medical and surgical facts relating to the etiology of hernia. We must recognize that medical and surgical facts permeate but slowly, especially when they have to overcome long-established traditions frequently supported by court decisions. The first requirement, in my opinion, is to convince the various commissions and courts, of the well-established surgical fact that hernia is a disease and not the result of an accident. When this has been thoroughly done, a radical review of the present state laws regarding compensation in cases of industrial hernia will be forthcoming. The active coöperation of the medical profession in this direction is absolutely essential. Board decisions and court decisions in almost every case are, in the final analysis, based on the medical and surgical testimony and opinion given at the time. If the profession generally accepts the opinion as expressed by the most eminent men in their own ranks as to the few instances of real traumatic hernia, it is most difficult to reconcile this with the testimony of many doctors when appearing before our boards. Recently a member of an industrial accident board in New England said to me, "I have read hundreds of pamphlets and books on hernia, and have come to believe that only a negligible number of them are properly compensable, but what can I do when in case after case on which I am compelled to sit as judge and jury, there is considerable medical and surgical testimony insisting that this particular case is the exception to the general rule."

In addition to the remedy above outlined, a recommendation of Dr. James W. Davis of North Carolina in "Southern Medicine and Surgery" in August, 1931 is of interest and importance. He says, "I feel that every employee should be asked to present to his employer before he starts work, a certificate from a reputable doctor that the employee has no hernia. A careful general examination should also be made, and a report of the result filed with the employer. I feel that the medical profession should adopt a standard fee for examinations of this kind, considerably less than the usual fee charged for a thorough examination. In all fairness to employees, employers, insurance companies, and industrial commissions I earnestly recommend that employees be required to file annually with their employers a certificate regarding the presence or absence of hernia." In support of this Dr. Hopkins in a paper on traumatic hernia reveals the important fact that of all the men who passed physical examinations prior to entering railroad service, the claims for traumatic hernia amounted to less than 1 per cent, while on the other hand 91 per cent of the cases of alleged traumatic hernia were found to occur in foreigners who did not have a preliminary physical examination before being admitted to service.

I firmly believe that if these indicated remedies are carried out consistently and persistently by all interested parties, that eventually and probably very soon, all states will include in their compensation laws specific provisions such as are at the present time in 22 state compensation laws in this country relative to the handling of claims for hernia.

It is perfectly clear that something should be done to stop the ever-increasing cost of workmen's compensation insurance, otherwise, as Mr. Jones has said, the system is likely to break down. Already some employers have ceased to become insured because of excessive cost. Certainly we are all very much interested in preserving this system. Employers of labor feel the burden the most and they will undoubtedly be the ones to sponsor the remedy, but they should be definitely and positively backed up in their efforts in this direction by enlightened labor leaders and the medical profession generally. Legislatures are bound to give careful consideration to any proposed law which in the final analysis is for the best interests of all the people. The result would be greater justice to both employers and employees and would do much to maintain the continuance of the workmen's compensation act, undoubtedly one of the greatest humanitarian measures ever enacted.

Discussion

Chairman Rice: The discussion will be opened by Dr. Herbert H. Howard of Boston.

DR. HERBERT H. HOWARD: I suppose some of you gentlemen wonder why a man who is interested in urology is here discussing a paper on industrial hernias. In 1917 I was asked by Dr. Donoghue, the medical adviser for the Industrial Accident Board, to make a study of the so-called industrial hernia in this State in order to see if some definite conclusions could be arrived at.

I have been very much interested in the subject ever since, but I am still in the dark as to just what constitutes an industrial hernia. The present chairman of the Industrial Accident Board, Mr. Joseph Parks, is doing everything in his power to get at the bottom of this problem, the expense of which is serious, not only to the insurance companies concerned, but, after all, to the employers in the State.

The main difficulties with the hernia question seem to be first, that each member of the Industrial Accident Board feels obliged to decide each case upon the evidence given at the particular hearing; secondly, the medical profession appears to be at sea as to what constitutes a hernia; and, lastly, the Industrial Accident law does not in any form or manner speak of hernia as an entity.

The Workmen's Compensation law is supposed to compensate a workman for any injury that arises out of and in the course of his employment.

It is well known that so-called traumatic hernias are almost unknown except for some penetrating wound. Therefore, the condition which we have to deal with is not the production of a hernia, but the aggravation of a hernial condition or a weak inguinal region.

If we could adopt the recommendation of the Committee on Public Relations of the Massachusetts Medical Society, that major operations, except as emergency measures, demand a consultation with the insurance company, a large part of the questionable hernias, of which there are many, would be thrown out of consideration.

The potential hernia, so-called, seems to be the refuge of the man who wants to operate whether or not there is a disability incident thereto.

I was very glad to be present when Dr. Moorhead read his paper.

I never have operated on a case of an industrial hernia and found any hemorrhage in the canal or any edema in the canal and I agree with him that such a condition does not exist in this type of case.

The report given out by one of the societies in New England to which Mr. Holland refers, was discussed by the Board, but as that report had to do with the production of hernias it did not do any real good in the administration of a law which calls for compensation for injury arising out of the employment.

Aggravation of a hernia to my mind requires immediate pain and, if the man does not complain of any pain until several days after the alleged accident, I feel quite positive that that hernia is not industrial and I do not see how anyone can say it is industrial, if they know anything about the anatomy of the inguinal region.

Mr. Holland's suggestion of having all the employees in the State examined before they are allowed to work is probably the only solution of the problem here in this State at the present time, but I believe that if the Board would measure the aggravation of a condition chargeable to the particular industrial happening we would be able to cut down the present expense in handling these cases and prevent many good workmen being thrown out of employment.

There is another situation which is rather difficult to solve and that is, the recurrent hernia. What constitutes a recurrent hernia, when the recurrent hernia is due to the original accident, when it is due to a second accident and how much time has to elapse between the two, the Board has not determined and there is no minimum or maximum limit at the present time. All recurrent hernias have a much longer, a much more prolonged disability than the fresh hernia. The expense to the company is much greater and it seems to me that something should be done soon to determine what constitutes a recurrent hernia.

If the Board would follow what appears to be good medical authority, that no injury produces a hernia and that the compensation is payable for the aggravation of a condition already existing, when that condition is remedied by an operation, the liability of that particular insurance carrier covering that particular employer should cease and if there is a so-called recurrence or a bulging of the scar later that results in a disability, it would be properly chargeable to the happening as of the time that it occurred and should not be chargeable back either to the hernia operation or to the congenital condition which the man had in the beginning.

If a man has a hernia and he knows he has had it for several years and he does some lifting today, and has a sudden pain which is not disabling and continues on with his work from that time on, I cannot believe that that man should come under the industrial act as it is in this Commonwealth. His aggravation is only a temporary affair and the hernia returns to the condition in which it was prior to this alleged lift.

If a man comes in with a scrotal hernia and on reduction the sac is found to be thick with

a large ring, it is reasonable to assume that that is not of recent origin and it cannot possibly have come down and attained that size and have a sac which is thick enough to be definitely diagnosed a thick sac. It is obvious that that hernia is of long duration. But still you will see these cases coming up quite frequently where they are diagnosed as fresh hernias.

I think this organization or the New England Surgical Society ought to determine upon some definition of hernia, agree upon some definition of what constitutes an aggravation of a hernia and when that aggravation ceases. Lastly, the medical profession needs education as to when the disability following an operation is at an end. If a patient who has had an appendix operation can go to work after three weeks, it does seem that warning given to laboring men by house-officers and many of the physicians that they should never engage in heavy work is rather far-fetched.

The importance of hernia is great enough that the surgeon at each hospital who has performed the operation should advise the man, and certainly after an operation the man is safer to go back to his employment than he has been in years before.

CHAIRMAN RICE: Dr. Edwin D. Gardner will continue the discussion.

DR. EDWIN D. GARDNER, New Bedford: Mr. Holland, so far as I know, has been here and heard the papers this afternoon and I think he must believe that all doctors are liars. We come here and we all agree that there is no such thing as a traumatic hernia, and then certain of us get up before an Industrial Accident Board and say that this hernia and that hernia are industrial—are traumatic. On the other hand, it has been my experience that doctors make this statement far less frequently than labor leaders and lawyers and patients.

A patient is asked if he got hurt. Yes. When? He tells the Commission. He says he had pain right off, and then he noticed a lump. And the Commissioner has decided then and there that it is a traumatic hernia, and it doesn't make any difference what a doctor says.

So far as feeling badly about the expense to the insurance companies, I can't particularly; because, after all—and particularly in mutual companies—the expense is borne by the employer and not by the insurance company. I feel very strongly, however, that this section of the Massachusetts Medical Society should take a very definite stand and say that there is no such thing as traumatic hernia and eliminate this term rather than continue all this argument which gets us nowhere.

I have been very much interested in seeing, since the period of depression, the great increase in the number of hernias which have come into the so-called public wards with no mention made of any accident. And, gentlemen, I feel very strongly that this thing should be stopped. I know that I feel guilty. I operate on these hernias, because they are sent to me. I don't get up before a commissioner or a judge and say that I believe that this or that hernia was traumatic. But I operate on them and, therefore, I believe I am guilty, and I believe that anybody else who operates on that type of hernia is guilty. I have never seen a so-called traumatic hernia—and I have seen a great many of them very shortly after the so-called injury—none of which showed any signs of trauma, either hematoma, swelling, tenderness or anything of the sort. It is quite evident in most of them that they have been present for a long time.

Mr. Holland made a suggestion that all employees be examined. It is perfectly true, that will do away with a certain number of cases, but I think that number will be surprisingly small because I believe that in a great many cases the hernia is so small, and in a great many cases there is no evidence of a sac. I know that in some plants where this method is followed there is case after case missed. That is no reflection on the doctor; you just can't find it. I have had cases come in to see me where some other doctor has said there was a hernia present, and I couldn't find a hernia except on three or four or more examinations. So that is not going to answer it.

I believe we must simply stand up and say there is no such thing. I think it makes no great difference, so far as hospital expenses are concerned, because somebody is going to pay, but it is a matter of right and wrong, not a matter of finances, and it certainly is wrong to say there is such a thing as traumatic hernia when there is no such thing.

CHAIRMAN RICE: Mr. Holland's paper is now open for general discussion.

DR. A. O. BERNSTEIN: I would like to ask Mr. Holland or Dr. Howard in regard to the suggestion of having all candidates for employment examined; would they have these candidates examined with the understanding that if a hernia was found they would be excluded from employment, or would they be accepted with that contingency? Secondly, if that is the case, what would organized labor feel toward this body for recommending such a move?

DR. F. L. NEWTON, Somerville: What is trauma? Much depends upon the definition. 1. Three men were throwing empty butter firkins up on the loft, and during the process one found he could not lift his arm. He had dislocated his shoulder. 2. Another found he had a broken bone in his arm from muscular contraction. The third felt a very sudden sharp pain down in the inguinal region and collapsed. The other fellows with their left arms helped

pick the third fellow up. Now, was that trauma? Were they subject to an indemnity from accident for injury? They all complained; they all carried accident insurance, and filed their claims and were each laid up for a length of time. The accident insurance companies recognized each one as having met with an accident and paid indemnity. 3. Accidents are not all traumatic. We doctors here know that. We have accidents that are not traumatic. There are accidents—as accidental hemorrhages. 4. Now, those that are recognized as accidents with injury receive compensation and are usually classed trauma. Trauma is defined broadly as "injury". It seems that the industrial insurance companies have gotten together in much the same manner as fire insurance companies and pay proportionately and there is a feeling that industrial insurance companies have combined a good deal in the same way. These systems of protection are not analogous. But the question is: is a man entitled to compensation for something which occurs to him while he is in employment and fulfilling his obligations in his occupation? There is a feeling that he is not so considered by protective insurance in many cases.

Now, previous to this we have spoken of, and a paper was read in regard to, "traumatic appendicitis". I think it is appropriate to this, too; it comes in under insurance. Suppose a man receives a blow in the loin, collapses, suffering apparently from concealed hemorrhages. Removal of the kidney reveals a large hard calculus. This was contributive. A young man has had several attacks of appendicitis, and in one attack called in a physician or surgeon, and an operation was advised and refused, he went away feeling he had recovered. So of this man the surgeon would know very well, though he saw through a glass darkly, that face to face, he would have found an active appendicitis. Soon after, through his occupation or athletic sports he gets a thump right over the seat of that appendix. He is a thin-walled boy, and he gets a good hard thump. It hurts him severely, and he collapses; he has trouble enough now, and his parents send for the surgeon, who operates and saves his life (if he doesn't do too much operating). That depends on his judgment. Some surgeons tell me that their judgment has done more for them sometimes than technique, and is responsible for more than fifty per cent of their success, which always has been good. That appendicitis is a disease, primarily, is beyond dispute—the terminal "itis"—signifying inflammation—determines this and that it originates in the lumen, etc., but that trauma through accident may be a secondary cause is possible and must be accepted as an individual condition.

Therefore, it is time to bring the conclusion down to the fact, as one man stated, to make it fair and just for the individual, in order that those cases may be rightly considered. You understand that I am not subsidized as an insurance surgeon. I think there is a subsidy, and I am saying it here flat and square, because I meet with those cases, and have never been willing to accept a case for the defendant under those conditions.

Therefore, I am speaking for the plaintiffs, the poor fellows that get hurt, and whom the insurance companies don't want to pay for the medical service. I just settled a case today. "Oh, no, we allow you two dollars." A man comes in with an infected finger. You operate on him. You see him every day, and dress his wound and look after it carefully. You get him well. You send in a bill for five dollars for the first visit and three dollars after that. They say, "No, we don't allow that. The Industrial Accident Board fixes the price. And you will get two dollars. But plenty of the men would pay you that difference." Some of my colleagues offset this by saying, "Social insurance and the doctor must contribute his part."

It is time, you say, to have something done. It is time when we come to a position where we look after the workers who earn the money that makes the millionaires. That is why it is time for us to do something, to give a chance to the men who get just enough to live on, while many of the millionaires in this depression pay no income taxes. It doesn't belong here; I know I am speaking out of order, but it is "all in the the same boat" with the subsidies of some of our surgeons. And I call it being subsidized when they all get up and talk on one side. It is not with desire to incriminate or to fail of appreciation but it is too apparent that bias is shown, that unfairness and injustice are practiced and the younger physicians are losing confidence. They are outspoken in their belief that "there is a reason". Has there been anything said here today with all these surgeons coming from different States in the interest of the plaintiff? No. Let him suffer. The first days when he is really suffering, he can't get paid. No, not until he has been disabled and out a certain length of time. Why?

I know I speak emphatically, and I speak with the experience of fifty years in practice. I have no personal complaint to make. I have been paid in almost every case I have had, if it didn't get into the courts. I find they are often more decent out of court than in. Go into the courts with the case, and get the poor fellow with a broken back, who lay on it for a year, with splints, with everything as clear in the case as can be that it was an accident and worthy of compensation—the insurance company regards it as advisable to make a good offer of settlement, but the attorney takes a chance, as he thinks, to make more by trial in court, puts him on the stand and makes a monkey of him. That also is a profession. The plaintiff's answer to a certain question in the

case that may call for a technical interpretation—a legal bi-play—the liability is not proved. It is as a matter of fact a just claim. There is nothing unfair in a general, or usual recognition, of associated conditions. But a fault in the phraseology—the use of a *word* in a statement as applied to an act, changes, in legal parlance, so that an honest plaintiff who would have received just compensation becomes a victim of injustice. In place of justice and righteousness—gets injustice and cussedness.

CHAIRMAN RICE: Is there any further discussion? Mr. Holland, would you like the opportunity to reply to your critics?

MR. JAMES H. HOLLAND: I have not heard any great amount of criticism, but I would like to answer a question that one of the gentlemen asked, what the situation would be following out the recommendation I made in regard to the physical examination of employees, the question being, "What would be the attitude of the employer as regards employing that man, and what would be the attitude of labor in regard to it?" Well, I am afraid I cannot answer that question. It certainly is not in the mind of any representative of any insurance company that I know of, because of such a physical examination disclosing the existence of a hernia, to bar that man from employment. The purpose of the examination would be to disclose the fact that he had one, and when that hernia eventually became disabling, to assist the insurance company and the Industrial Accident Board in barring him from recovering for a condition not due to an industrial accident. There is no intention in the mind of anybody in the insurance field to prevent the man from getting employment.

CHAIRMAN RICE: Dr. Marble, I apologize for not giving you an opportunity to reply to the discussion.

DR. HENRY C. MARBLE: In the matter of hernias I feel that all of us are talking about different things. We all admit that in the matter of traumatic hernias the patient probably had a hernial sac before the injury. I think that no doctor believes that a new hernial sac is formed and driven down into the scrotum by trauma. As the result of a lift or a strain or any undue exercise the preëxisting hernial sac is filled. I do not believe that there is any question about this. It is entirely a question of aggravation or acceleration of a preëxisting known condition.

Now I want to say a few words about the matter of traumatic appendicitis which Dr. Newton has brought up. I cannot believe that trauma to the abdomen or any blow has anything whatever to do with the cause or aggravation of appendicitis. This is an entirely different matter from that of hernias and I cannot let the remark of Dr. Newton go unchallenged.

CHAIRMAN RICE: The Secretary has a letter which perhaps requires some action by this section.

June 5, 1933.

Dr. Ernest M. Daland, Secretary,
Section of Surgery,
Massachusetts Medical Society.

Dear Dr. Daland:

Next week the Committee on Public Health is to take part in a publicity campaign for the earlier recognition of appendicitis. This campaign is to be patterned on that of the Philadelphia County Medical Society two years ago, and is being entered into jointly by us with the State Department of Health, the Massachusetts Pharmaceutical Society, and the Boston Retail Druggists Association.

Cards for distribution by the Fellows of our Society are obtainable at the registration desk during this meeting.

Although individual members of your Section have been consulted during the preparation of our material, we would greatly appreciate your endorsement of the project. Merely for your convenience I would suggest some such form as follows:

Resolved: That the Section of Surgery hereby endorses and approves the campaign for the earlier recognition of acute appendicitis which is being projected by the Committee on Public Health of the Massachusetts Medical Society, The Massachusetts State Department of Health, The Massachusetts Pharmaceutical Society and the Boston Retail Druggists Association.

Faithfully yours,
DWIGHT O'HARA,
Chairman, Committee on Public Health.

CHAIRMAN RICE: What is your pleasure in regard to this resolution? Would somebody move that we endorse it?

(*Voted*, that the resolution be endorsed.)

CHAIRMAN RICE: Is the Nominating Committee ready to report?

DR. CLUTE: The Nominating Committee proposes the name of Dr. Ernest M. Daland for chairman of the Surgical Section for the year, and Dr. Joseph H. Shortell for Secretary of the Section.

(*Voted*, that the report of the Nominating Committee be accepted.)

CHAIRMAN RICE: Is there any other business? Gentlemen, we have had two men speak to us this afternoon, one from out of town and one from outside the profession. It seems to me that in carrying the motion to adjourn it would be proper and courteous to extend to Dr. Moorhead and to Mr. Holland a rising vote of thanks.

(With a rising vote of thanks to Dr. Moorhead and Mr. Holland, the Section adjourned at 4.30 P.M.)

The attendance at this Section was 250.

THE NEW HAMPSHIRE MEDICAL SOCIETY

MEDICAL CARE OF THE COUNTY POOR*

BY JOHN P. BOWLER, M.D., HANOVER†

IT does not require that one be a student of public affairs to have acquired during the economic developments of the past three years a consciousness of the presence of increasing problems in the work of the physician and increasing pitfalls for the harmony and prestige of our membership. One may recognize as well the persistence of the traditional laissez faire attitude of our professional group which is most directly concerned with some of the larger factors in these problems, an attitude which, if allowed to continue, will in time remove our organization from the position of a participant in the discussion of a solution which must be found.

Within our State the care of the poor without established residence falls within the jurisdiction of the County Commissioners, of whose total work this field now constitutes some 75 per cent, and which is rapidly increasing at the present time by virtue of the ruling which transfers dependent cases from town to county responsibility after three years of unpaid poll taxes. As a result of the increase in unemployment and county dependents, county budgets have reached a point at which, as a fiscal operation, they far outrun the possibilities of the existing machinery, created as it was under the conditions of a far different day. It is not our intention to enter the discussion of the efficiency of our county system of government, as to whether the office of a county commissioner should be an elective one, whether its functions should be uncontrolled to the present extent, or whether it should involve the possibilities of patronage. For whether the system remains in its present form, or is changed by legislative enactment, or is forced to give way to State intervention as a means of avoiding county bankruptcies, the problem of medical care of the county poor will still exist as a direct problem of the medical profession and as an opportunity on its part for the offering of a real contribution.

The problem of the members of a county society is limited to the off-the-farm county poor. The county farm is an institution under the control of the County Commissioners and county physician. The recent tremendous increases creating the present problem are in the off-the-farm cases scattered over the counties. The amount of money necessary in meeting these increasing loads of the counties is presenting a problem now well known. Increasing budgets to be raised by taxation with tax collections decreasing have already impaired the credit of some counties to the point of virtual bankruptcy. An item of not inconsiderable size in these expenditures is represented by medical care and, under the present entire absence of system, lends itself probably least of all items to budgetary control.

On inquiry through letter to the secretary of each county society, in no county was there found a definite fee schedule for pauper cases or any machinery by which such cases were handled. Fees ranged from total charity to normal average charges which led in most cases to the comments that there was general dissatisfaction with the situation, inconsistency leading to unfair competition and ill-will within the profession, a feeling of unduly increasing imposition on free service, the opportunity for bartering between commissioners and physicians, and the general conclusion that some action should be taken. It is to be admitted that this total lack of any uniform schedule has resulted in a few instances in such impositions in the rendering of fees that there has been set up in the minds of some county authorities the generalization that the entire profession represents a group from which little coöperation can be expected. In so far as there are grounds for this attitude, the fault lies definitely in the inaction of such medical organizations as our own.

A perfectly human reaction on the part of an official charged with such public responsibilities is to get as much as possible for as little as possible. This has led to the accumulation of county work in such hospitals as provide ward service free of professional charges. At the same time, some county hospitals have been allowed to use their accommodations for private patients at times to the extent that it definitely interfered with their primary and, rightly, sole function of the care of public dependents. Such use of county hospitals has no justification. Existing as they do, entirely on taxation revenue, their operation in competition with institutions supported by voluntary public charity and needing the income from private patients to aid in balancing less-than-cost rates to many patients is obviously open to serious question of function.

It was on this general background and in the belief that the county society represents the most efficient unit for such procedures that the Grafton County Medical Society discussed and unanimously adopted at its meeting this month the Grafton County and Town Pauper Fee Schedule for Professional Services. The schedule was based generally on that already estab-

*Read at the Annual Meeting of the New Hampshire Medical Society at Manchester, N. H., May 17, 1933.

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lished by the physicians of Lebanon as an agreement between them and the local authorities. Briefly, this schedule was adopted as an attempt at the relief of the dissatisfaction with existing conditions and trends, as a fair reconciliation of the increasing demands on the physician and his obligation in such an emergency, and for the continuation of the handling of the local patient by his local physician. This schedule was purposely drawn up in broad general classifications rather than in detailed itemization covering all possible contingencies for the sake of brevity and conciseness and to allow scope for the application of the judgment of the individual physician. Rulings as to necessary authorization for such professional services by the public officials involved were obviously necessary to allow control on the part of the officials responsible and to prevent that which is termed by them as unnecessary pauperization of some individuals unwilling to accept that status, merely because they are unable temporarily to meet their medical costs. The schedule as adopted is as follows:

1. Office Visits—$1.00.
2. House Visits—
   A. $1.50 within 2-mile radius of office;
      15¢ mileage charge for each mile traveled outside of the 2-mile radius.
   B. Home obstetrical cases — $15.00, covering total services.
3. Hospitalized Cases—
   A. Medical cases—$1.00 per day.
   B. Surgical cases — $3.00 per day.
   C. Obstetrical cases—$5.00 per case.

Maximum fee on any case within any one accident or illness, $100.00.

Fees for special work in connection with county pauper cases such as special laboratory work (outside of that provided by State service), anesthesia, etc., shall be rendered at 50% of the standard rate.

A Committee was created by vote of the Society at this meeting consisting of the County Physician, a member of the Society elected for two years, and the President of the Society as chairman. This Committee will operate as the County and Town Schedule Committee, to whom is to be referred any protest by a member of the County Society or the County Commissioners relative to any matter concerned in the operation of this schedule. This should eliminate the necessity of any discussion between the County Commissioners or their representatives and the physician concerned in any case.

This schedule is adopted by the members of the Grafton County Medical Society and the Commissioners of Grafton County under the following agreement:

1. That the operation of this Schedule as beginning on June 1, 1933, shall render void any existing agreements in any locality relative to the carrying out of this work.
2. That the County Hospital shall limit its activities to the hospitalization and care of county and town pauper cases.
3. That beyond a first emergency visit no additional procedures by any physician on any case shall be carried out without authorization from a County Commissioner or his representative to whom a report must be made within forty-eight hours of such first visit; that otherwise no obligations shall be assumed by the Commissioners.

An interesting commentary on this procedure in Grafton County was our finding it necessary to pass a motion for suspension of the rules of the organization in order that we might adopt this schedule. It might be presumed that this program could be criticized as a step in the direction of state medicine. Assuming that term to mean physicians in the employ of the state, no more definite step in the opposite direction could be taken than this plan which proposes to leave the local patient in the care of his local physician. Since the writing of this report the New Hampshire Legislature has passed Bill No. 417 by which the State takes over the administration of all poor relief. While subject to rejection or modification by the State Senate, this recent development makes the theme of this discussion all the more pertinent.

As has been well said, a thesis might be written on what is happening to the medical profession in the absence of its own planning to meet the exigencies of a radically changing society. This program as adopted by Grafton County is, we believe, a step in the initiating on the part of the profession itself a plan for meeting a definite emergency which from the standpoint of all parties concerned requires some definite action.

Discussion

Dr. Fred Ellsworth Clow, Wolfeboro: If out of the present depression the medical profession in this state does not emerge with a clear understanding on the part of doctors and laymen alike that *Society is responsible for its own* we will have proved ourselves incapable of cohesive action for the safeguarding of interests that are our own, by all the standards of modern life. So long as the state recognizes its duty to provide for all its people, who are unable to procure for themselves, food and shelter, we should by sturdy and steady pressure require that medical service be classed as a necessity, and so considered for adequate remuneration. No longer should the doctor be forced to bear the burden alone. Our medical societies should stand firmly on this platform because it is only by united action that we can establish our rights. Too much in the past we have as organizations held ourselves aloof from demanding what is logically ours. The welfare of the patient has always preceded any thought

of the welfare of the physician. At last, even our national body has come to the conclusion that there is such a phase of medical practice as medical economics. The customs of another day when the doctor could do business and be grateful, if and when the patient pleased himself to offer recompense, are gone and buried under the stiff, competitive, complex struggle of 1933.

It is to the credit of the county for which Dr. Bowler speaks that a group of physicians has taken this matter in hand and set an example for the rest of us, for our own salvation, to follow. Some years ago we found to our chagrin that the constitution of our county society forbade the arrangement of a uniform fee table to apply to a homogeneous unit of population such as our little corner of the state. We have long maintained that these points should be clearly borne in mind:

1. The individual physician can do little by himself. Officials can drown his voice, no matter how earnestly he pleads. He is sure to be the loser in his efforts to get officials interested in just two matters: a low tax rate and a continuance of their administration, to accept responsibility that is properly theirs. Too frequently the individual doctor has found that vague promises mean only repudiation, and that the action of these officials is little short of plain dishonesty.

2. The smaller the community the worse the doctor's chances to get officials to agree to pay for the care of the poor.

3. The worst cases the doctor meets are those on the borderline between pauperism and independence. These are the ones who frequently refuse aid, quiet the doctor's demands with empty promises, and have their conduct abetted and condoned by officials who thereby are saving money.

4. The county or municipal hospital has no more right to demand free attendance by its medical staff than it has to require purveyors of food and clothing to do the same. Altogether too long physicians have been tempted by the prestige that such appointments were supposed to carry. Under the system that we now know, the free service given annually by the medical staff in the city and county hospitals of this country can be reckoned in millions of dollars. Until a few weeks ago no one had ever even thought to give credit in an annual report for such work, and Dr. Goldwater's suggestion is only a timid one.

5. County commissioners never before had so much money to spend for the care of the poor. Everybody expects high taxes. The public is in a mood to consider very leniently large expenditures of money for public welfare. And this situation will continue for many years. Thousands of those now receiving public aid will never be removed from this dole, no matter how general business improves. Once they become accustomed to look to society either for work or a dole there will never be again, for them, any other alternative. In addition, conservative economists estimate an unemployed population of eight million, in future, as a result of technological unemployment.

6. The legal distinction between what makes a man a town pauper or a county pauper is a relic of the past. The mere fact that there is bound up in this question the possibility of divided responsibility between town officers and county commissioners has increased the dilemma of the doctor who is often in need of the most direct action. In any event, the cost of the pauper's care comes from one source: taxation, —city, county, or state,—and of which, please be reminded, the same doctor is paying his honest share. Hope is held out to us that House Bill 417 will mark the dumping of legislation, serviceable in oxcart days, but decidedly outmoded in the days of the aeroplane.

Last year I sent, in connection with a piece of work done for a national committee on medical economics, a questionnaire to two hundred doctors in towns of four thousand and less in Maine, Vermont and New Hampshire. The last question was this: "Do authorities make payment for the care of county poor, outside hospitals?" Over one hundred physicians replied to this, giving free comment. Apparently our sister states have better arrangements in certain respects than does ours. But a note of discouragement, and dissatisfaction, and lack of morale ran through them all. Over seventy complained most bitterly of the borderline case, where it is easily possible for the officials to evade responsibility.

It is interesting to note that it is not only in this part of the country that arrangements such as Dr. Bowler has outlined are being made, but such states as Michigan and some counties in Wisconsin, are setting up such plans. Parts of Iowa have had such contracts for twenty-five years, certainly long enough to prove their feasibility. Populous Suffolk County, Long Island, has solved its problem of the care of the poor as well as the independent. Of these schemes Van Etten says, "All over the United States county societies are actively discussing —formulating and administering medical service plans of many kinds; this is as it should be. Something valuable will be solved in these laboratories."

Two considerations must govern our conduct:

1. Costs of service must be kept at a reasonable level. Justifiable criticism has been directed at our actions in the past. Surgery and expensive laboratory work must be kept at a minimum. Patients must be treated at home often to the doctor's inconvenience.

2. A large measure of patience and conciliation must characterize our attitude in dealing with men whose chief interest will always be to save money rather than to see eye to eye with us.

Our sincere gratitude is due Dr. Bowler and his committee for calling emphatic attention to this important phase of medical practice. We look to the House of Delegates to make such pronouncement as will insure the county societies adequate backing in the negotiations that will follow. Is it too much to hope that the influence of our powerful national organization will be on the side of the overburdened family doctor in his attempt to secure for the present, and safeguard for the future, his own rights, at a time when he is suffering the worst exploitation he has ever known? For the first time in its history medicine is on the defensive, largely as a result of our own inaction in the fuller development of the social implications of our profession. The future depends upon our own ability to measure up to the requirements of a new age, to new ideals, to new social obligations.

DR. EDWARD R. B. MCGEE, Berlin: Dr. Bowler has given us a very learned paper, one which shows considerable care and ability in its preparation and I heartily agree with him on every point. There is nothing much that I can add to what he has already said, but at this time I would like to bring up how this matter is handled in Coos County, especially in Gorham and in Berlin, where I am most familiar with the workings of the County Commissioner's office.

If a family needs a home visit from a physician, a district nurse is sent, first to discover whether the home call and the expense of a physician are necessary. The family is asked if they have a regular family physician and if so, he is sent if possible. If not, the visits are distributed equally among the various physicians. If an operation is necessary and it is an acute case, the patient is admitted to St. Louis Hospital. If the case is a chronic one, the patient is hospitalized in West Stewartstown at the County Hospital.

The physicians of Berlin and Gorham agree during the present financial crisis to amend their fee schedule for county cases, for one year as follows:

Office visit	$ 1.00
House call	2.00
House visit after 7 P.M.	3.00
Consultant fee	3.00
House visit outside City Limits, 50¢ a mile, plus	2.00
Chemical analysis of urine	1.00
Obstetrics	15.00
Fractures, ordinary	15.00
Major operations	50.00
Throat operations	15.00
Minor operations, as amputation of finger or toe, including assistant's fee	15.00

Since November 1, there has been an outpatient clinic at the St. Louis Hospital which is open to anyone in Berlin who has not sufficient income to patronize a physician, whether or not he is receiving aid from public funds. The admission desk is controlled by the office of the social worker and her assistants, who check up on the financial income and decide whether they should be admitted. A general medical and surgical clinic is held on Mondays and Wednesdays at 8:30 to 9:30, and the eye, nose and throat on Tuesdays. The general clinics are held by a different doctor each month. All of the doctors give their services but receive pay for any operation which comes during their period of service at the clinic at which diagnosis has been made. The towns in the other parts of the county, like Lancaster and Whitefield, Colebrook and Stewartstown use a local physician, but of course in the latter vicinity, the county hospital is used to a greater extent.

In conclusion I would suggest that during these distressing times of economy and depression, it would be advisable for the physicians in the different counties, as suggested by Dr. Bowler to come to some definite agreement as to standard fees and to coöperate with their respective commissioners in an effort to carry out these plans. In times past the physician was considered quite an important factor in the functioning of the affairs of his community, but of late years he has gradually drifted away from all civic activities due to the many increasing duties of his profession. It would seem that he should retrospect a bit and once again take a real active part in the political situation of his city, state and nation, as there is no one more fitted to represent the common people, than the family doctor who comes in daily contact with his patients in their homes and everyday life and who is so thoroughly familiar with their needs.

By so doing I feel positively sure that all physicians will be able to keep in closer touch not only with the affairs of their communities but also with the many things pertaining to their profession and can work advantageously for the best interests of their many colleagues.

DR. D. E. SULLIVAN, Concord: For the benefit of the membership I think it should be stated that the House of Delegates has unanimously endorsed the recommendation contained in this paper of Dr. Bowler, and has asked the delegates from the different county societies to report that action to their county societies at the very first meeting following this, and that each

County Society get busy in carrying into effect the plan given by Dr. Bowler. To my mind, the medical profession has been the tool of all sorts of agencies for too many years. Perhaps this has been due largely to its own inaction but this is a most opportune time for the profession to insist upon and demand its just rights. The Constitution of our land provides for food, shelter, raiment and health, and they are all paid for by taxation except health, which is most neglected. It has been expected that the profession would work without recompense. Now is the time, to stand up and be men and exact the consideration we are entitled to. The solution of the problem is in your own hands. Certainly our asking for compensation for services rendered is nothing but our just right in the matter. And I think you will find that we shall have the backing of the officials of the State and the people in general in this matter. And so, men, when you return home, go to it! I believe you have never been called upon as a profession to assert yourselves as you are at this particular moment.

DR. JOHN P. BOWLER, Hanover: I would like to make one or two things clear. We went at this problem from the point of view of a local situation on the basis that, in the absence of any system, it had led either in actuality or at least in potentiality to a good deal of ill-will in the group because of the inconsistency. On that basis, it seemed as though something ought to be worked out. The other matter is the action in the Legislature, by which if this all goes into organization under the government or the council, if we have nothing in the picture at this time, if we have no claim, then it is obvious that in any discussion we are simply not recognized and that is the main point.

I think that in the discussion it has been emphasized that there is opposition to the idea on the part of county officials. I think that isn't so. We found that not at all in our county. In fact, they are very well pleased that a rule under which they can operate is in the picture.

DR. ROBERT J. GRAVES, Concord: There isn't much more to say on the subject. My investigations were in Merrimack County, and from these investigations, it seems to me that educating physicians is fully as important as educating the Commissioners. The experiences of the physicians, particularly in small towns, have led them to believe that the present system, with the absence of red tape, was quite a good deal more workable than the proposed one. But I doubt very much if there are any of those who cannot be sold on the subject when it is properly presented to them.

DR. ALFRED T. BAZIN of Montreal: *Mr. President, and Members of the New Hampshire Medical Society*—This takes me rather unawares, and I don't know but that before the day is completed you will be tired of hearing my voice, inasmuch as I come on the program officially this afternoon and officially also this evening.

In addition to saying how glad I am to be here and how I feel honored to be invited to this State meeting, I gathered from the few remarks I heard since being in this room during the last few moments that you are wrestling with a problem of the medical care of the indigent. Your problem is not confined to yourselves. Every country, at the present time, has that problem, or ought to have it. I might almost say that the organized medical profession in every country has that problem, and more particularly ought to have it. The medical men of our country are supposed to be intelligent. They are citizens. As citizens they ought to appreciate their responsibility to the country. As intelligent citizens, they ought to be able to guide, and, in a formative way consider the problems that devolve upon the citizens, and more particularly they ought to be able to guide the solution of problems that refer to the health of the community.

Now, I don't know just what your specific problem may be, but in Canada, it has been accentuated during these past three years of economic depression. Our Federal government has been willing to assist financially our nine provincial governments for the provision of relief to the unemployed on the basis of a contribution from the federal government of fifty per cent of the total required, the other fifty per cent being found by the provincial and the municipal governments. We, of the organized medical profession in Canada, were awakened to the fact that the Federal government was not prepared to extend financial relief except for the provision of food, housing, fuel and clothing. It apparently did not come into their purview that health was also important, the reason for that being that under the British North America Act, which was the Act constituting the formation of the Dominion of Canada, the provinces were allowed, or at least they insisted upon retaining all their rights in regard to education, elementary, secondary and professional. Custom has decreed that "Health" is also the prerogative of the provincial governments.

Organized medicine in Canada has corrected that erroneous impression in the provinces and in the whole country, and the federal government is now willing to permit the fund to be used for health, too. It has been left to the provinces individually to consider the details of the plan by which they will relieve the indigent sick.

The medical profession is proud of its traditions in caring for the sick who are unable to pay, but they are proud of that tradition inasmuch as it has always been a privilege and a voluntary contribution. But we feel that as a profession we should not be *obliged* to care for the indigent sick when for every other need

of the indigent person the government has some way of providing funds.

So, in certain of the provinces, this is the scheme that has been put into effect. Realizing that it will cost the government a great deal more to hospitalize a sick individual than to treat that case at home in the incipient stages of the illness, the profession is permitted to charge for the treatment at home of those who are so afflicted. The accounts are then sent to the Relief Officers of the District. The doctor knows of course, when he is called to see a patient, that that patient or the family of the patient receives relief, and so he sends his account to the Relief Officer of that District, and the government, coördinating with the Medical Society who agreed to this plan, out of the relief fund pays fifty per cent of that account, the medical man, through organized medicine, agreeing that the other fifty per cent is his contribution to the economic disaster of the times.

Now, in order to safeguard the government and the funds against exploitation, it is also agreed that no matter how much a doctor attends families or individuals on relief, the total amount he can get from the government in any one month is one hundred dollars. That is a scheme that is working out very satisfactorily.

I am quite ignorant of what your exact problem is. But that is one way that certain provinces in Canada have overcome the difficulty. We think that this is a very fair way of doing it, not only to the medical profession but to the government and its available funds.

THE SECONDARY NODULES OF LYMPHATIC TISSUE*

Their Relation To Immunity

BY RALPH E. MILLER,† HANOVER

THE extreme importance of the reticulo-endothelial system in relation to immunity is evidenced by the frequent appearance in the recent literature of treatises upon various phases of research in that field. The fact that the importance of the research is appreciated in few clinical fields is evidenced by the scarcity of correlating clinical studies.

One conclusion that may be drawn from a review of the literature in the field of cytology, histology and pathology of the lymphatic system, is that the conflicts of opinion expressed in interpreting the observations may more probably be answered by clinico-pathological studies rather than by too narrow investigations of the purely laboratory side of the question. The lymphatic system is a large part of the reticulo-endothelial system, and the availability of tonsils, appendix and lymph nodes for study by the clinician, histologist and pathologist is an open invitation to make worth while advances. So long as the lesions of the lymphatic tissue are all included under the heads of hyperplasia, neoplasia, and inflammatory conditions, without consideration of the parts of the system involved, little more can be done concerning a solution of the intricate problems involved.

Lymphatic tissue (Aschoff) is composed of two distinct anatomical elements, parenchyma and nodules. Whether these anatomically different elements are an expression of a physiological difference remains to be proved. The evidence in favor of such a physiological difference has been accumulating since Brücke first described the secondary nodules of lymphatic tissue in 1850. It is proposed here to record the salient features of the accumulated evidence with the hope of stimulating an apparently dormant clinical interest in those structures characteristic of lymphatic tissue which may well prove to be as essential to existence as many of the more dramatically exploited portions of the body mechanism.

Except for the generally, though not universally, admitted scavenging function of lymph nodes (Herring and MacNaughton) (a function which subepithelial lymphatic tissue of the alimentary tract does not have, because it has no afferent lymph vessels), there is no agreement concerning the function of lymphatic tissue apart from its participation in the reticulo-endothelial system.

It is surprising that the earliest recorded attempt to correlate the structures peculiar to lymphatic tissue (the secondary nodules, germ centers of Flemming) with immunization was as late as 1899 by Manfredi[1]. Although Good[2] suggested again that the immunizing function of lymphatic tissue was its chief one, and Digby[3] independently over a number of years (1911-19) developed such a thesis, the profession generally has not accepted the idea. Recently the studies of Kaiser[4] of Cleveland have stimulated the clinical interest in the function of those parts of the lymphatic tissue.

Histologists and pathologists have contributed much in recent years to our knowledge of the reticulo-endothelial system (Aschoff, Kiono, Jordan, Higgins, Foot and many others) and not least of the phases of that study have been those

*The Trustees of the New Hampshire Medical Society awarded second prize to Dr. Miller for this essay.

†Miller—Assistant Professor of Pathology, Dartmouth Medical School. For record and address of author see "This Week's Issue," page 607.

related to the possible immunizing function of the system (Nicolle and Conseil, Digby, Dudley). The so-called "block" experiments by which the reticulo-endothelial system is supposedly deprived of activity by filling its cells with dye particles are strongly suggestive, though inconclusive because of the tremendous regenerative power of the reticulo-endothelial cells and the extreme difficulty of determining whether the whole system is blocked. Some of these experiments have indicated a definite immunizing activity of the reticulo-endothelial system.

Many indirect experimental attacks designed to unravel the problems involved in a study of the lymphatic system have been made recently to show that the secondary nodules (germ centers of Flemming) play an important part in the changes which occur in lymph nodes, spleen, and subepithelial lymphatic tissue under varying circumstances (Hellman[6], Miller[5], Ehrich[7]). Miller recently summarized those findings with the conclusion that the earlier contention that the secondary nodules were chiefly places for lymphocyte formation must give place to the idea that the secondary nodules have primarily some other function.

The summaries of Kaiser and of Digby and particularly those researches of Hellman and of Heiberg, of Heilman, of Ehrich and of Miller point directly to the association of the secondary nodules to a probable immunizing function of lymphatic tissue.

The evidence here recorded supports the thesis that the secondary nodules of lymphatic tissue originate, develop and regress according to the demands of the bodily economy for detoxification of foreign substance, whether they are bacteria, disintegrating tissue, or circulating soluble substances.

Much of the evidence is circumstantial and therefore must be considered as only suggestive; but it points definitely to the conclusion supported by experimental data and clinical experience.

No clearer expression of the part played by the sub-epithelial part of the lymphatic tissue can be given than that of Digby[3], many of whose conjectures have been borne out by recent experiments.

He states:

"The subepithelial lymphatic glands confer upon their owners some immunity against the alien myriads of bacteria. They are immunizing stations, or, if you like, the auto-vaccination organs of the body." He proceeds to summarize the evidence, as it refers to the subepithelial lymphatic tissue.

The biological evidence points to the lymphatic system with secondary nodules as a late phylogenetic development, it being limited to birds and mammals. Their late development and persistence would indicate the opposite of their being a vestigial, non-functioning organ system.

Lymphoid tissue is almost universally present in the mammalian cecum, and sometimes this forms a protuberant mass at the apex. Digby says in support of his contention that the appendix is a functioning lymphatic organ. "Quite frequently and in many widely different phyla the lumen of the caecal apex is small. The lymphoid tissue then thickens the wall of this tubular mechanism. The maximum amount of lymphoid tissue is associated with the tubular type and occurs in rodents where the caecum is of immense size and serves for the bacterial decomposition of cellulose."

The secondary nodules are the functioning elements of lymphatic tissue, in their relation to immunity. Throughout the body the distribution of the lymphatic tissue containing secondary nodules is understandable only if we consider that the nodules have some protective relationship. The popliteal nodes of many rabbits are marked by larger and more numerous secondary nodules than the inguinal. The mesenteric lymph nodes in carcinoma contain few secondary nodules of the active type unless the first line of defense, the subepithelial lymphatic nodules, has been destroyed.

The secondary nodules of the alimentary and respiratory tract are separated from the lumen of the organ by a single epithelial layer and it is in these subepithelial nodules, where exposure to organisms is almost constant, that the secondary nodules develop first and persist longest.

Wherever there is stasis of the gut contents lymphatic tissue with secondary nodules is abundant and in many places such as the tonsillar crypts, the appendix and cecum, there is definite anatomical provision for stasis and the consequent opportunity for juxtaposition of secondary nodules and intestinal contents. But where stasis exists in the absence of bacteria secondary nodules are not found in abundance, for instance, in the stomach. If infection, irritation or a disturbance of the normal relations occurs in regions where nodules are not usually found such as the nasal mucosa, urethra, ureter, bladder and even in the kidney, beneath the epithelial covering typical lymphatic tissue with secondary nodules may be found.

Physiologically the formation, persistence and regression of secondary nodules of lymphatic tissue follow so closely the natural association of man with bacteria that one must suppose some relationship. Digby found constantly in rabbits' appendices bacteria within the lymphocytes and macrophages. It is reasonable to assume that the same occurs in human lymphatic tissue. There is some parallel evidence for human lymph nodes reported by Herring and Mac-

Naughton[8]. Bunting and Huston[28] found that the lumen of the rabbit's intestine contains a number of lymphocytes greater than the number contained in the blood stream and they account for the loss of 3,300,000,000 lymphocytes from the blood stream each day by this route.

We must believe that the constant ingestion of bacteria leads to some development of antibodies when we consider all the evidence of immunology. It is not coincidence that mere exposure to disease may result in immunization, without development of the clinical disease, and that the hypertrophy of tonsils in children is precisely at the age when they are coming in contact with many new organisms. The old age regression of tonsils and appendix and lymph nodes is parallel in its significance because of the constantly decreasing contact with new organisms. That the presence of secondary nodules of the active (germ center) type is the distinguishing characteristic of that lymphatic tissue which is exposed to the prolonged, mild action of bacteria (or their secondary effects) was pointed out by Ehrich[9], and Miller[5] reported the same from long-standing chronic inflammatory changes in tissue, whether bacterial in origin or not.

Ehrich[10] demonstrated a cycle of development of the secondary nodules in the popliteal node of the rabbit following abscess development in the extremity distal to the node. Miller[5] reported the almost constant relation of secondary nodules in the lymph nodes draining gastric ulcers, benign and malignant, in contrast to the scarcity of the secondary nodules in the gastric lymph nodes of a larger control group of comparable age. It is the common knowledge of physicians and surgeons that lymphatic tissue atrophies with age, and it might be inferred that this is due to decrease in contact with *new* organisms, particularly in the light of the findings of Miller in gastric ulcer and the common experience of surgeons and pathologists that the lymphatic tissue of elderly people responds to the presence of cancer (or other chronically destructive lesions) by the development of secondary nodules of the active type. The studies of Hellman[6] and others indicate that the hypertrophy of lymph nodes and their subsequent atrophy is dependent in great part upon the changes in number and size of the secondary nodules rather than other parts of the lymphatic tissue.

Sabin's[11] and Ehrich's[12] studies of the embryology of lymphatic tissue, and the observations of many others are one of the strongest bits of evidence (though circumstantial) that the secondary nodules of lymphatic tissue are directly related to the contact of the animal with bacteria. A recent thorough review of the literature on the subject of the development of the secondary nodules indicates general agreement that only the primary nodule (without germ center) is developed in utero and that the development of the secondary nodules is associated with the contacts of extra-uterine life. Gulland examined fetuses of man, rabbit, sheep and guinea-pig and found no "germinal centers". Baum reported that he and his students had not been able to find germinal centers in horse, pig or dog before the fifth day of life, nor in the cow before the eleventh day. Gundobin found, on investigating lymph nodes of 50 newborn infants and 100 children from one to two years of age, that the first germinal centers appeared at the age of two months, and that typical, well-formed germinal centers appeared at two years of age. Jolly and Rosello found that the spleen of rats did not contain germinal centers up to three weeks of age, and that the centers did not reach full development until two months of age. Goslar, on investigating 34 human tonsils from stillborn infants, living infants and adults, concluded that "germinal centers appear relatively late in extra-uterine life". Parodi, in a study of the lymph nodes of 66 human beings varying from 35 cm. fetuses to adults of 85 years found no germinal centers in the fetuses and rarely in persons more than 30 years of age. Ehrich studied chiefly the axillary lymph nodes of 48 fetuses and newborn infants, varying from 14 to 40 weeks in utero, and found secondary nodules in a single case. This was in the spleen of a 34 cm. embryo, the mother of which had died of pneumonia. Hellman was unable to find secondary nodules in the tonsils of rabbits before birth. Collins found no secondary nodules in an examination of 50 fetal appendices. An even stronger suggestion of the possible immunizing function of the secondary nodules is indicated by the recorded instances (Hellman), (Margerstedt[13]) of the finding of the secondary nodules in the lymphatic tissue of fetuses born of diseased mothers. From these experiments one is compelled to the conclusion that the primary lymphatic nodules (capillary tufts surrounded by lymphocytes) are the fetal anlage of a later functioning organ, the secondary nodule. The concept, originally advanced by Flemming and later supported by many, that this later developing secondary nodule is a specialized place for lymphocyte formation appears disposed of by the observation that lymphocytes appear in the body long before secondary nodules appear and lymphocytes are formed by lymphatic tissue long after the regression of the secondary nodules. The work of Downey and Weidenreich[14] demonstrated the abundant formation of lymphocytes outside the secondary nodules.

Maximow's[15] description of the cyclic changes in the secondary nodules considered in the light of Ehrich's[12] studies of the correlation of such changes with abscess formation at the same time

that lymphocytosis regressed indicates some other function than lymphocytopoiesis for the secondary nodules.

Hellman's[6] designation of these nodules as reaction centers indicates his conviction that they have some other function than lymphocytopoiesis and his reticence to assign to them any such definite function as immunization centers, indicates a state of mind common to most workers who have attempted to solve the nature of these definite nodules found only in lymphatic tissue under certain circumstances associated with tissue destruction or bacterial infection of low intensity.

It might be argued from the block experiments that the secondary nodules do not participate in immunity production because, in a blocked animal, immunization is slowed and in such animals the cells of the secondary nodules do not contain the dye. It is probable that the secondary nodule is merely one of the links in the immunizing chain and that the blocked reticulo-endothelial cells of the surrounding parenchyma are another and therefore the blocking though incomplete, is adequate to interfere with immunity production.

Digby's[16] observations of the rabbit's appendix indicate some such partial participation of the secondary nodule. He found lymphocytes apparently migrating into the lumen of the appendix, ingesting bacteria, then returning to the lymph nodule where they were apparently acted upon and disposed of. A similar activity of phagocytes acting to carry bacteria to lymph nodes where they are acted upon was found by Herring and MacNaughton[8]. Clark and Clark[17] demonstrated a similar activity for fat droplets. These authors' works demonstrated that lymphatic endothelium itself has the power of active collection of foreign material into the lymphatic stream.

Digby is much quoted in references to studies of the function of the tonsils, and he deserves commendation for forcefully and repeatedly presenting to an apparently skeptical profession ideas which have recently been substantiated in a clinical analysis along lines which he suggested in these words, "What is required is to take many thousands of healthy children living under similar conditions, in half of whom the healthy tonsils have been enucleated and the adenoids completely cut off within the first two or three years of life, and then to compare the susceptibility of the two groups to infections during the remaining years of childhood."

In 1930, Kaiser[4], in a much quoted work, reported results of observations on 2,200 tonsillectomized children with an equal number of controls. The conditions were not so ideal as those specified by Digby, but the results are so thoroughly in accord with the findings in other clinical studies of smaller series[18, 19] that they must be considered as contributing a decided advance to our knowledge of the value of tonsillectomy. Of more fundamental importance, however, is the clinical support of the theories of the immunizing function of lymphatic tissue. This is certainly an argument for the preservation of any lymphatic tissue which is known definitely not to be diseased. All those who practice medicine and surgery upon the upper respiratory tract must have realized experiences like that of Kaiser and of Greenbaum[19], and there is a noticeable reticence on the part of many to remove tonsils unnecessarily. This reticence might well be practiced when other lymphatic organs are the point of attack. The increase in the incidence of respiratory diseases (pneumonia, bronchitis, laryngitis) found by Kaiser in the tonsillectomized group, if borne out by future series on large groups, can mean only that the tonsils are a definite defense organ. The arguments brought forward here make it reasonable to apply the conclusions to the lymphatic system as a whole, and though Nussbaum[20] after a very complete review of the field stated that the function of the tonsils had never been demonstrated, it would appear unnecessary to await a *direct* proof before acting upon the mass of evidence in favor of that function being a protective one.

It must be assumed, from the study made by Wilkinson[21], of 10,000 pairs of tonsils that all tonsils after the first few days of life have crypts containing leucocytes, débris and bacteria; that this condition so far as the body economy is concerned must be a normal one.

Like all others the lymphatic organs are subject to disease and the very ease with which they are accessible to pathogenic organisms makes them the site of serious affections. This does not mitigate against the conclusion that they are protective organs. All must be familiar with the acquiring of immunity by some in scarlet fever epidemics by having a mild tonsillitis, and every physician has seen the course of a local infection proceed to swelling of the neighboring lymph node followed by regression of the lesion (without surgical intervention) but persistence for a time of the swollen node. Ehrich's[10] work, quoted above, in which he found the persistent lymph node swelling, after staphylococcus abscess formation, due to an increase in the size and number of secondary nodules of the active type, suggests the part played by the secondary nodules.

The negative evidence for protective function of the secondary nodules is the frequent finding of an absence of active secondary nodules in persons dying of fulminating infections. There is not a failure of the whole lymphatic system in such instances, because there are plenty

of circulating lymphocytes, and lymph nodes may appear normal except for the absence of the secondary nodules of the active type. The necrotic type of secondary nodule occurs so frequently in severe diphtheria that it is considered characteristic of that disease, and this necrosis may well be considered as evidence of the breaking down of the immunizing system.

The function of the secondary lymphatic nodules of the spleen has been correlated to that of the other lymphatic tissue by the work of Ehrich[22] who injected killed staphylococci intravenously into rabbits and produced an increase in the size and number of the secondary nodules of the spleen. It might be concluded that the spleen acts for the blood stream in a manner similar to that of the secondary nodules of lymph nodes for the rest of the body.

That the exact manner in which protection is brought about by the lymphatic tissue still remains obscure should not prevent our acting upon the knowledge that it plays some active part in immunization.

The evidence from purely pathological studies is also circumstantial but points to the same conclusion; that is, the very susceptibility of lymphatic tissue to invasion by bacteria is a provision for immunity development. The lymphatic tissue of the intestine is the site of first attack by the pathogenic organisms of the colon-typhoid group and upon their integrity depends the eventual outcome.

The argument brought forward here should in no way be construed to prevent resection of lymphatic tissue whenever and wherever there are indications for that procedure. The purpose of this paper is to correlate the abundant clinical, physiological, embryological, histological and pathological records to the end that the importance of the little understood secondary nodules of lymphatic tissue may be realized.

SUMMARY

1. There is need for clinical-pathological correlation of the data accumulated from various fields of investigation of the lymphatic system.

2. No function has been proved as that of the secondary nodules of lymphatic tissue.

3. When all available evidence is assembled it becomes apparent that the structures peculiar to lymphatic tissue, the secondary nodules (lightly staining central parts of lymphatic nodules, which normally contain many mitotic figures) bear a relationship, physiologically, pathologically, anatomically, and embryologically to the external world, which makes them logically organs for the protection of the host against foreign materials.

a. Secondary nodules appear only after the individual's contact with external influences.

b. They are not necessary for lymphocyte production.

c. They are situated in those places best suited to exposure to bacterial action.

d. They increase and decrease in size and number directly according to the severity and variety of infection.

4. Reports of clinical analyses of series of tonsillectomized and untonsillectomized children indicate that the possession of tonsils is definite insurance against respiratory infections.

5. Experimental and clinical data make it apparent that this protective action demonstrated for tonsils is applicable to other lymphatic tissue.

6. It is proposed that there is sufficient evidence to support the belief that lymphatic tissue has a definite function associated with immunization and that the secondary nodules of lymphatic tissue are an integral and essential part of this mechanism.

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MISCELLANY

RECENT DEATHS

EASTMAN, EUGENE B. — With great regret it becomes my painful duty to record the sudden death of Eugene B. Eastman, M.D., of Portsmouth, who died on September 6, 1933. The cause of death was hypertensive heart disease and chronic nephritis.

His education was gained in the University of New Hampshire, Tufts College Medical School, and McGill University of Montreal, Canada.

His great ability early earned him prominence in his chosen profession and his work was known far and wide by its sterling quality.

His friends were legion, both within and without the profession and people in all walks of life were benefited by his contact.

A quiet, unassuming man, Dr. Eastman went his daily way and many people in straitened circumstances felt his philanthropy. His many deeds of kindness can never be enumerated, and countless ones will never be known excepting in the hearts of those whom be befriended.

No task was too small or too large to receive his undivided attention. The difficult situations that he met and overcome are too numerous to comment upon.

A man among men ever ready to battle for what he believed to be right, a beloved brother to his colleagues whom he always stood ready to help at any time of the day or night, a friend to the weak, a guiding hand to the strong and a beloved counsellor whose advice was sought far and near; his place will never be filled in the hearts of those privileged to love him.

He was a member of the New Hampshire Medical Society and the Massachusetts Medical Society. He occupied the position of trustee of the New Hampshire State Hospital. The Portsmouth Hospital early gave him a position on the staff which institution he loyally served for many years. He served as medical referee of Rockingham County for a number of years.

He was a Mason for many years, having taken all the degrees on each road, and in this he always evidenced deep interest.

He was married in Portsmouth on December 19, 1905, to Annie J. Hill. The home life of this couple has left nothing to be desired and to those who knew them no further comment is necessary.

Dr. Eastman is survived by his widow, one brother, Charles, and one sister, Mrs. L. A. Robeck, of this city.

WENDELL P. CLARE.

ATHERTON—ELLA BLAYLOCK ATHERTON, M.D., of 31 Fairmount Street, Nashua, New Hampshire (office at 168 Main Street), died in that city September 4, 1933. She was born in Ulverston, Lancashire, England, January 4, 1860, and moved to Canada at the age of three. After her premedical education at McGill University, she entered the Queen's University Faculty of Medicine and was given her M.D. degree in 1887. Prejudice against women physicians existing in Canada prompted her to open an office in Newport, Vermont, and after two years she moved to Nashua where she practiced surgery. She was a member of the New Hampshire Medical Society and the American Medical Association.

MEMBERSHIP CHANGES

NEW MEMBERS

John L. Norris, M.D., New London. Graduated from McGill University Medical School 1931. Licensed in 1933.

Dr. William Dennis Thibodeau, 226 Main Street, Nashua.

Dr. Walter George Wroblewski, 174 Main Street, Nashua.

RESIGNED

Dr. Alfred A. Macleay, 814 Elm Street, Manchester.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending September 2, indicate a mortality rate of 9.6 as against a rate of 9.9 for the corresponding week of last year. The highest rate (17.9) appears for Washington, D. C., and the lowest (4.4) for Flint, Mich. The highest infant mortality rate (13.7) appears for Oklahoma City, Okla., and the lowest for Albany, N. Y., Lowell, Mass., Lynn, Mass., New Haven, Conn., Portland, Oreg., Schenectady, N. Y., Waterbury, Conn., and Yonkers, N. Y., which reported no infant mortality.

The annual rate for 85 cities is 11.0 for the thirty-five weeks of 1933, as against a rate of 11.4 for the corresponding period of the previous year.

SUMMARY OF DEATHS AND DEATH RATES (ANNUAL BASIS) FROM AUTOMOBILE ACCIDENTS PER 100,000 ESTIMATED POPULATION FOR 86 CITIES FOR CORRESPONDING PERIODS OF 1933 AND 1932

	Week ending Sept. 2, 1933	Week ending Sept. 3, 1932	First 35 weeks 1933	First 35 weeks 1932
Total deaths	176	147	4,975	5,007
Death rate	24.5	20.9	19.8	20.3
Deaths due to accidents in city	137	109	3,917	4,046
Death rate	19.1	15.5	15.6	16.4

—*Bulletin Bureau of the Census.*

CLINICAL AND ECONOMIC FEATURES OF ARTHRITIS IN EX-MEMBERS OF THE MILITARY SERVICE*

BY PHILIP B. MATZ, M.D.†

(Continued from page 554, issue of September 14)

FOCAL INFECTION

Millard Smith[7], in a study of a group of 102 cases of atrophic arthritis, found that the cause or causes of the arthritic condition at times were subtle and diverse so that each patient must be studied individually in order to discern the etiological factor, as well as the succession of events which precede the inception of the arthritic process. This observer found that patients were rarely seen in whom a single condition could be pointed out as the etiological factor.

Thirty-seven per cent of the group studied by Smith gave a history of a preceding illness; all but three of the conditions were of an infectious nature.

A number of observers maintain that one must not adhere too rigidly to the theory that focal infection is the etiological factor. Infection acts as an excitant, but it may be preceded by certain predisposing factors. In the group of atrophic arthritis studied by Smith, 50 per cent gave such histories. These predisposing factors were of a varied kind and consisted of physical, nervous, mental, or profound emotional traumata.

Pemberton[1] maintains that the theory of the part played by focal infection in the causation of arthritis has been carried to extreme and that the pendulum of medical opinion is now swinging toward the mean point. He further holds that it is still impossible to offer precise information as to when a focal infection is or is not responsible for systemic effects. This observer holds that arthritis may follow attacks of ill health of many kinds, even periods of fatigue, worry or physical strain, and these may act as predisposing factors.

In a study of a group of 400 arthritics in the army hospitals during the World War, Pemberton noted that the most frequent precipitating factor leading to the inception of the joint disease was exposure to cold or wet; it played a leading rôle in nearly 80 per cent of the cases of arthritis.

A study of the type of focal infections causing arthritis has resulted in the opinion that the principal foci are in the teeth, tonsils, or the genito-urinary tract. Frequently more than one focus is recognizable. The present study confirms these observations.

The question may be asked how the organisms produce arthritic lesions and why the joint is the site of the phenomenon called arthritis. Arthritis is not always accompanied by the presence of microörganisms, inflammation, and pus formation, at least a study of the joint fluid fails to show these manifestations in many cases. Then, too, it is difficult to establish a relationship between the bone changes accompanying the several forms of arthritis with specific organisms. Such bone changes may be explained upon the basis of a chemical transformation rather than a process due to bacterial activity.

The phenomenon of arthritis may also be explained upon the basis of an allergic mechanism of a bacterial type, the joint manifestation merely being a tissue response to a bacterial antigen operating in the focus of infection. As in tuberculosis, allergy is an index of the degree of immunity against infection with the tubercle bacillus, so in arthritis, the joint changes may be said to constitute an allergic manifestation indicating the degree of immunity against bacterial action elsewhere in the body.

Zinsser, who is one of the leading exponents of the theory that arthritis is an allergic manifestation, is of the opinion that it is difficult to believe the disease is caused by any particular bacterium, but that the phenomenon may be the result of the activity of various kinds of organisms resulting in joint-tissue response.

While the nature of the etiological factors, as well as the mechanism involved in the inception of the joint disease, that of bacterial allergy, concern particularly the atrophic type, nevertheless arguments may be adduced to show that cases of hypertrophic arthritis may give a history of similar causative precursors, and there is a possibility of a common pathology and abnormal physiology similar to the findings in the atrophic variety of joint disease, so that an arbitrary identification of the joint affection as atrophic or hypertrophic is at times not justifiable.

Pemberton[1] in a study of a group of 400 arthritics in the army, found that 293, or 73 per cent, gave evidence of a focal infection. This same observer, in a study of a group of 163 subjects affected with arthritis, found that 126, or 77 per cent, showed the presence of a focal infection.

Miller[12] states that there is abundant clinical evidence to show that atrophic arthritis is infective in character. This type of joint disease may appear with an acute onset, and may be accompanied by fever, leukocytosis, inflammation of the joint, and enlarged spleen and lymphatic glands. Rosenow, as well as Billings, Coleman and Hibbs have obtained positive cul-

*From the Research Subdivision, Medical and Hospital Service, Veterans' Administration.

Presented at the Conference on Rheumatic Diseases, held under the sponsorship of the American Committee for the Control of Rheumatism, New Orleans, May 9, 1932.

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tures from the regional lymph nodes of cases of atrophic arthritis. Cecil, Nicholls and Stainsby obtained positive streptococcus cultures from the blood in 61.5 per cent of a group of arthritic patients. Other investigators have recovered staphylococci from the joints of arthritic patients.

Miller[12] is of the opinion that the focus from which the bacteria reach the blood stream and finally the articular surface of the bone is frequently indeterminable. Some observers emphasize especially abnormalities of the digestive or of the urinary tract, others the teeth and tonsils, still others are of the opinion that acute nasopharyngeal or tracheal infections, rather than chronic infections are responsible for the joint involvement. According to Miller the nature of hypertrophic arthritis is such as to indicate a noninfective etiology. Many theories have been advanced as to the etiologic agencies that might explain degeneration of cartilage, which is the outstanding pathologic finding. Fisher is of the opinion that hypertrophic arthritis is a physiological response to some form of irritation either mechanical or chemical. Key refers to instances in which acute trauma of joints has followed disturbance of posture leading to faulty weight-bearing or strain.

Mixed forms of atrophic and hypertrophic arthritis may be explained upon the basis of long-continued mild trauma, age, and strain, as etiological factors of the hypertrophic arthritis, which generally follow the occurrence of atrophic arthritis.

Wetherby and Clawson[13] found the frequent occurrence of streptococci in the blood stream of a group of 57 patients with various clinical forms of chronic arthritis strongly suggesting an etiologic relationship.

Nichols and Richardson[14] are of the opinion that atrophic arthritis is not produced by a single etiological factor but is the result of a great variety of irritants most of which continue to act for a long time. In some cases the active cause can be discovered, in others the cause is unknown and can only be suspected. These observers maintain that the joints of atrophic arthritis do not start clinically as an acute process. On the contrary, they often start insidiously, with no acute symptoms, progress with a number of remissions and continue in many instances for a number of years. If these joint lesions were due to bacterial infection it is probable that the disease would acquire immunity and come to a spontaneous end. Moreover, the clinical symptoms and tissue changes are not those of an acute bacterial infection, but are suggestive of chronic irritation with localization in the joints. The initial changes are in the synovial membrane and consist of a proliferative process. Another assumed cause of the proliferative joint changes is an irritant, the result of faulty metabolism, which for some unexplained reason selects the joint cavities and epiphyses of the bones as the sites of most marked activity. The causes of hypertrophic arthritis, according to Nichols and Richardson, are conditions which expose the joint to severe strain or long-continued pressure over localized areas, which result in degeneration and fibrillation of cartilage. Such pathological lesions of the joint cartilage may occur in chronic organic disease of the central nervous system, as for example, tabes dorsalis. It may be the result of exposure to severe mechanical action in abnormal positions, as, for example, in hallux valgus, or in dislocations.

The data of the group of 450 arthritics which constitute the material of this study indicate that 298, or 66.2 per cent, gave evidence of one or more infective foci to which the arthritic condition might be attributable; 82.6 per cent of the group of arthritis of the atrophic type, and 52.6 per cent of the group of arthritis of the hypertrophic type, gave a history of a focal infection. Arthritis of the spine and pelvic joints, both of the hypertrophic and atrophic varieties, also gave evidence of a focal infection.

While it is conceded that atrophic arthritis may be due to infective foci, the presence of such foci in hypertrophic and other types of arthritis, to the extent shown in this study, leads one to infer that infections may possibly play a part in the causation of the arthritides other than those of the atrophic variety.

SITES OF FOCAL INFECTIONS

Glover[4] states that by far the most frequent sites of the septic focus in cases of chronic arthritis are the teeth and gums. Willcox, in a study of a group of 100 cases of chronic arthritis, fibrositis, and subacute rheumatism, found the frequency of focal infection as follows: dental sepsis 72; intestinal infection 13; tonsillar sepsis ten; and urethral infection five. Cecil and Archer, in an analysis of 200 cases of chronic atrophic arthritis, found that infected tonsils constituted the most common foci; these occurred either alone or in combination in 61 per cent of the cases; the next most common foci were in the teeth, either alone or in combination, 33 per cent; the sinuses, prostate, gall bladder, colon, and cervix also provided foci of infection.

Of the group of 450 cases of arthritis in this study, 298 gave a history of a focal infection to which the joint disease might be attributed. The most frequent sites of the infective process causing atrophic or hypertrophic arthritis as well as combined hypertrophic and atrophic arthritis, were the gums and teeth, tonsils, nose and throat, genito-urinary tract, sinuses and intestinal tract; influenza and pneumonia were also etiological factors of some of the cases included in the study.

In the case of arthritis due to specific agents,

the genito-urinary tract was the most frequent site of the focal infection. In cases of rheumatic fever, the most frequent antecedent infections were in the tonsils.

It has been previously stated that the focal infections to which the arthritis was attributable in some instances occurred singly, while in others in combination. The most frequent single infections which were followed by arthritis were those of the gums and teeth, tonsils, influenza, intestinal tract, etc. The most frequent combination of focal infections prior to the inception of arthritis was of the gums and teeth and tonsils; this combination occurred in 50 instances.

MODE OF ONSET

The mode of onset of the joint disease is dependent upon the type of arthritis. Fisher[5] states that in atrophic arthritis every stage may be encountered between the acute types and the more chronic and afebrile variety of less sudden onset, which may merge into the hypertrophic form of arthritis. The onset of hypertrophic arthritis is usually insidious and may be associated with slight symptoms so that the patient is not conscious of the joint involvement. Of the group of 450 arthritics comprising this study, in 148, or 32.9 per cent, the onset was acute, and in 302, or 67.1 per cent, it was gradual and insidious. It was further noted that in 26.3 per cent of the group of hypertrophic arthritis, the onset was acute. In 64 per cent of the group of atrophic arthritis the onset was gradual.

CHANGE OF WEIGHT DURING COURSE OF ARTHRITIS

Chronic arthritis is usually associated with a marked change of weight. In the majority of instances a loss of weight is noted. This is said to be due to the presence of toxins in the arthritic process, together with a nutritional disturbance which frequently accompanies the joint disease.

Smith[11] in a study of 102 cases of atrophic arthritis, observed an appreciable loss of weight during the course of the disease.

Three hundred and seventy-seven of the group included in this study were reviewed for a change of weight during the course of arthritis. It was found that 255, or 67.7 per cent, gave evidence of a loss of weight following the onset of joint disease. The largest group, 93 in number, lost between 10 and 20 pounds, however this loss was gradual and extended over a period of years. Ninety-one, or 24.1 per cent, showed a gain in weight following the onset of arthritis. The largest group, 40 in number, gained between one and ten pounds. This gain however extended over a period of years.

LABORATORY EXAMINATIONS

Urinalysis: Smith[8], in a study of 102 cases of atrophic arthritis, found but two in which the urinalysis showed the presence of albumin. Pemberton[1], in a series of 700 civilian arthritics, found six cases of diabetes of a mild type, an incidence of 0.85 per cent.

In the group of 450 ex-service arthritics, it was found that 41, or 9.1 per cent gave evidence of albuminuria and 34, or 7.6 per cent, showed the presence of casts. It was found that 12, or 2.7 per cent, of the arthritics had a coexisting nephritis.

Four, or 0.8 per cent, of the 450 arthritics showed evidence of glycosuria. It was noted that but one of the group had a coexisting true diabetes mellitus.

Leucocytes: Glover[4], in a study of a group of arthritics, found leucocytosis present in those with atrophic arthritis; a marked leucocytosis was found in cases of hypertrophic arthritis; and moderate leucocytosis was present in cases of gout.

Munro, in a study of the blood of arthritics, found that 57 per cent of the cases showed a leucocytosis varying from 9000 to 20,000. Hypertrophic arthritis showed a relatively larger per cent of leucocytosis.

In the study of a group of 118 of the ex-service cases with hypertrophic arthritis, it was found that the total leucocyte count ranged from 4700 to 16,200; the average was 8120 per cmm.

In 134 cases of atrophic arthritis the total leucocyte count ranged from 4550 to 24,200; the average was 9045 per cmm.

In 16 cases of hypertrophic arthritis of the spine and pelvic joints the leucocytes ranged from 4700 to 13,100; the average was 8365 per cmm.

Among six cases of arthritis due to specific agents, the leucocyte count ranged from 6150 to 12,800; the average was 9558 per cmm.

Among 8 cases of rheumatic fever the leucocytes ranged from 4200 to 28,500; the average was 10,650 per cmm.

Considering the group of 290 arthritics, in which blood findings were recorded, the range was from 4200 to 28,500; the average was 8678 leucocytes per cmm.

From the data obtained in this study one may state that, generally speaking, chronic arthritis is not accompanied by a leucocytosis except in specific instances. The highest leucocyte counts were present in cases of rheumatic fever and the next highest counts in cases of atrophic arthritis.

Erythrocytes: It is generally agreed that atrophic arthritis is accompanied by a slight secondary anemia. Hypertrophic arthritis, however, is not associated with an anemia.

Pemberton[1] in a study of the erythrocyte count in a series of 339 arthritics, found that 32 per cent showed a count of less than 4,500,000

per cmm. Smith[8] in a study of a group of 46 patients with atrophic arthritis, found the usual incidence of secondary anemia which other writers have reported. No particular investigation was made by this observer to determine the cause of the anemia, but it was his opinion that it could be accounted for by the profound toxemia from continued focal infection, or from marked debilitation with resulting inadequate dietary.

In the study of 111 cases of hypertrophic arthritis among ex-service men, the erythrocyte count ranged from 3,800,000 to 6,950,000; the average was 4,859,207 per cmm.

In 132 cases of atrophic arthritis the erythrocyte count range was from 3,600,000 to 5,850,000; the average was 4,731,686 per cmm.

In 16 cases of hypertrophic arthritis of the spine and pelvic joints the erythrocyte count varied from 4,030,000 to 6,500,000; the average was 4,672,750 per cmm.

In the consideration of the whole group of 279 patients with different types of arthritis, the erythrocyte count varied from 3,600,000 to 6,950,000; the average was 4,779,952 per cmm. It was also found that but 3.9 per cent of the total number of 279 arthritics had an erythrocyte count of less than 4,000,000.

It is thus seen that in the group of ex-service men with chronic arthritis the joint disease was not generally accompanied by a secondary anemia. This is not in accord with the findings of other observers.

Hemoglobin: In a group of 279 ex-service men with arthritis, the hemoglobin per cent varied from 60 to 110 per cent; the average was 84 per cent. It is of interest to note that 18.3 per cent of the total number of 279 cases showed less than 80 per cent of hemoglobin. This is in accord with the findings of Pemberton[1] who, in a study of 289 cases of arthritis, found that 20 per cent of the group had less than 81 per cent of hemoglobin, the latter being considered normal.

Blood sugar: Pemberton[1] noted that there was a definite relationship between the amount of carbohydrate intake and the incidence and perpetuation of arthritis in a considerable percentage of the cases. Furthermore, this observer found that in arthritis a reduced carbohydrate tolerance was frequently seen and that the tolerance returned to normal with improvement of the joint disease.

Snyder and Traeger[15] in a survey of 1200 cases of arthritis, found that a great number of the patients had high blood sugar. In a group of 425 arthritics in office practice, these observers found that 136, or 32 per cent, had blood sugar values of 120 mgm. or more. Of another group of 732 clinic patients with arthritis 278, or 32 per cent, had blood sugar values above 120 mgm. None of the patients proved to be diabetic. Snyder and Traeger noted that as treatment progressed and the symptoms cleared up, the blood sugar values tended to approach normal.

In a study of a group of 186 ex-service men with arthritis, the blood sugar was above 130 mgm. per 100 cc. in eight, or 4.3 per cent, of the cases. The highest blood sugar content, that of 167 mgm., was found in a case of hypertrophic arthritis; the lowest blood sugar content, that of 50 mgm., was seen in a case of atrophic arthritis. The mean blood sugar content was 97.2 mgm. per 100 cc.

Uric acid: Glover states that uric acid of the blood is increased in practically every case of gout and in many cases of hypertrophic arthritis. J. Race found that there was a marked increase of uric acid of the blood in 79 per cent of cases of hypertrophic arthritis. Maizels and Payne found the uric acid of the blood abnormally high in eight out of 15 cases of atrophic arthritis investigated at Guy's Hospital in 1927. In a series of 731 cases of arthritis seen in clinic practice, Snyder and Traeger[15] found that 381, or more than 50 per cent, showed a decided increase in blood uric acid levels, in spite of the fact that more than 90 per cent of these patients had been on dietary restrictions with especial reference to meat. In a series of 425 arthritics in office practice 250, or more than 59 per cent, showed an increase of the blood uric acid.

Folin and Denis found a high uric acid content of the blood in arthritis. Chace, Myers and Killian failed to find high blood uric acid values in true arthritis when the kidneys were not involved, and believe that when higher than normal values are present it is due to impaired kidney function rather than to an arthritic process.

In a study of a group of 155 ex-service arthritics, the highest uric acid content was 8.9 mgm. per 100 cc.; the lowest uric acid content was 1.1 mgm.; and the mean content was 3.6 mgm. per 100 cc. It was further noted that 82 per cent of the group showed a uric acid content between two and five mgm. per 100 cc. and 12.8 per cent of the cases gave evidence of a uric acid content in excess of five mgm. Accordingly, one is justified in saying that arthritis may be accompanied by a slight increase of the uric acid content of the blood.

Non-protein nitrogen: Pemberton[1] found that the non-protein nitrogen and urea nitrogen values of the blood in cases of arthritis were within normal limits in the majority of instances.

In a study of the blood in a series of 194 ex-service arthritics it was found that 27, or 14 per cent, showed a non-protein nitrogen content in excess of 40 mgm. per 100 cc. The lowest non-protein nitrogen content of the blood

was 20.7 mgm.; the highest was 63.1 mgm.; and the mean non-protein nitrogen content was 33.1 mgm. per 100 cc. Accordingly, one may say that on the whole, arthritis results in a slight increase of the non-protein nitrogen content of the blood.

Creatinin: In a study of the creatinin of the blood in 190 ex-service men affected with arthritis, it was found that the lowest creatinin content was 0.98 mgm.; the highest was 2.5 mgm.; and the average was 1.48 mgm. per 100 cc. These figures indicate that the creatinin content of the blood in the arthritides is within normal limits.

Calcium: With the advent of newer quantitative methods for the determination of calcium in small amounts of blood, a number of investigators began the study of the calcium of the blood in arthritis. The conclusion reached by these workers was that there was no increase of the calcium of the serum or whole blood in joint disease.

Weil and Guillaumin, in 11 cases of chronic arthritis, reported serum calcium values ranging from 11.1 to 17.8 mgm. as compared with the normal of nine to 11 mgm. per 100 cc. Watchorn also found increased serum calcium in chronic arthritis, as did Horwitz in a series of cases of arthritis with marked deformity.

Cajori and Crouter, quoted by Pemberton[1], found that the serum calcium in a group of arthritics was within normal limits. There was no indication of any tendency toward high serum calcium in even the severe or chronic cases. Pemberton states that whereas there is an undoubted local disturbance of calcium metabolism in the immediate neighborhood of joints showing marked osseous change, this disturbance is not reflected in the circulating blood.

In a study of a group of 164 ex-service men affected with arthritis, 72 per cent showed a normal calcium content of the blood, i.e., between nine and 13 mgm. The highest calcium content was 17 mgm.; the lowest was six mgm.; and the mean calcium content of the blood in the group of 164 cases representing the various types of arthritis was 10.6 mgm. per 100 cc. It is of interest to note that the mean calcium content of five cases of rheumatic fever, 8.5 mgm. per 100 cc. of blood, was lower than that of any other type of arthritis.

Complement fixation test: Pemberton[1], in a study of 400 arthritics in the military service, found that eight, or two per cent, gave a positive Wassermann test. In these cases the luetic infection seemed to have no etiological relationship to the arthritis.

Of a group of 425 ex-service men with arthritis 12, or 2.8 per cent, showed the presence of a positive complement fixation test. Of these 12 cases, syphilis was the etiological factor of the arthritic process in but two instances.

A complete list of references will appear at the end of this article, which is to be continued.

MANY CAUSTIC POISONS INADEQUATELY LABELED

A survey by the Baltimore station, Federal Food and Drug Administration, of poisonous chemical compounds, including lye, carbolic acid, and oxalic acid, sold by dealers in the District of Columbia, indicates that a material proportion of these substances now on sale do not comply with the provisions of the Federal caustic poison act, according to W. G. Campbell, Chief of the Food and Drug Administration. Results of this survey, said Mr. Campbell, logically suggest that many retail dealers in other cities are selling caustic or corrosive substances without labels showing their dangerous character.

Retail dealers in many cities buy such products as carbolic acid, oxalic acid, hydrochloric acid, and silver nitrate in bulk and deal the products out at retail in small quantities. The health interests of the public, Mr. Campbell said, demand that before such sales are made the poisons be clearly labeled as to their dangerous character.

The two important requirements of the Federal act are placing the word "Poison" on the label in letters of a specified size and style, and a statement of antidotal treatments in case of accidental personal injury. The caustic poisons and the preparations containing them in the specified concentrations, which by law must be labeled, are silver nitrate, carbolic, oxalic, hydrochloric, sulphuric, nitric, acetic, and hypochlorous acids, and ammonium, sodium, and potassium hydroxides. Many of these substances are commonly used in the household for cleaning, disinfecting or other purposes. All of them are potentially injurious to the body if not carefully employed.

Early this year inspectors of the Food and Drug Administration visited druggists, paint dealers, and other retailers in the District of Columbia and informed them as to the labeling requirements of the Federal caustic poison act with respect to labeling the chemicals which come within the law, Mr. Campbell stated. Later, in order to determine the effect of this educational effort, official samples of caustic poisons were obtained from 85 dealers. Examinations of these samples showed that 58 of them were either not labeled in compliance with the caustic poison act or carried no labels at all. A large proportion of the carbolic acid samples were not labeled to comply with the requirements of the Federal food and drugs, insecticide, and caustic poison acts. Carbolic acid and many other caustic substances are virulent poisons. Routine preliminary hearings, required under the Federal laws, are now being held in Washington to permit druggists and other dealers to show cause why their cases should not be referred to the Department of Justice for prosecution. — *Bulletin U. S. Department of Agriculture.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19381

JAUNDICE AND BILIARY COLIC WITH ENLARGED LIVER

SURGICAL DEPARTMENT

PRESENTATION OF CASE

DR. CHAMP LYONS:* This case is that of a seventy-seven year old Italian, an unemployed fruit vender.

His first record, at the Pondville Hospital, four years prior to this entry, showed that he had had jaundice of three months' duration. He was discharged without treatment and instructed to report. He failed to do this. Two months later he was visited by a social service worker and was found to be free from jaundice and working as a fruit vender.

There is no further record from that time to the time he entered this hospital. His family knew little of the details and he spoke very little English and did not want to transmit any information. There was a history of alcoholism in early youth. He was nauseated and vomited on three occasions. For five months or more he had had rare attacks of colicky right upper quadrant pain, not radiating, and for the same period jaundice which had greatly deepened. He had never had a chill. The urine had been dark and the stools light. There was no itching of the skin.

On admission he was noted to be an extremely emaciated, markedly jaundiced man with moderate tenderness in the right upper quadrant. The icteric index was 100. A liver function test showed 100 per cent retention. A van den Bergh was 25.53, direct reaction. The urine was dark, the stools clay colored. There was a mass taken to be liver palpable in the right upper quadrant and extending two finger-breadths below the costal margin. The edge was felt. No evidence of fluid was made out in the abdomen. The white cell count was 14,000, the red cell count 4,460,000.

A gastro-intestinal series showed no deformity of the stomach or duodenum. A flat plate failed to reveal any calcified areas in the region of the gall bladder.

DR. GEORGE W. HOLMES: The film confirms the physical findings. There is no evidence of anything abnormal other than the area of dullness in the right upper quadrant, which is consistent with a large liver. There is no evidence of fluid.

DR. LYONS: He was followed in the ward by icteric index and van den Bergh tests taken every other day, with no change. The night after entry and again the following night he had an attack of severe right upper quadrant pain lasting about half an hour, associated with spasm and very marked tenderness in both upper quadrants. It was decided to operate in the hope that a diagnosis of obstructing stone in the common duct could be made. Three days after operation he died.

*Senior interne on the West Surgical service.

CLINICAL DISCUSSION

DR. LYONS: At the time of his stay in the Pondville Hospital he was seen by Dr. Henry Jackson, who made a diagnosis of carcinoma of the liver.

DR. GEORGE A. LELAND: This case presented a picture of a man presumably in the throes of his last illness. The story which we got through the Pondville Hospital of intermittency of his jaundice with acute attacks of pain prior to his admission here suggested very strongly that he had either mud or multiple stones in his common duct. It was on that basis that we did an exploratory operation in the hope that we should immediately find a large common duct which we could open and drain. It seemed to be his only chance. If we did not find that, we felt that exploration would shorten his existence by some weeks.

When we operated we found a small gall bladder and a small, normal common duct. We dissected down very carefully to make sure there was nothing there. The pancreas showed nothing, nor did the sphincter of Oddi. The liver was enlarged. Bearing in mind the diagnosis of primary carcinoma that Dr. Henry Jackson had made we carefully palpated the whole of the liver, and remarked at the time that perhaps there was something in the central portion of the liver, but we could not feel it. We took out a specimen for microscopic examination and made a postoperative diagnosis of biliary cirrhosis. From that time the patient did not recover full consciousness from the anesthetic, which was ether, and in thirty-six hours died.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Obstructive jaundice.

ANATOMIC DIAGNOSES

Carcinoma of the stomach with metastases to the liver.
Operation wound, biopsy of the liver.
Biliary cirrhosis of the liver.
Jaundice.
Bronchopneumonia.
Arteriosclerosis, moderate, aortic, coronary and renal.
Perisplenitis.
Chronic fibrous pleuritis, left.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: The small piece of liver which Dr. Leland removed for biopsy showed a fairly marked typical biliary cirrhosis, which of course ordinarily occurs only when there is obstruction or prolonged infection of the biliary tract. They had not been able to find any at operation and we found at autopsy no obstruction outside the liver itself.

The liver was enlarged, perfectly normal in shape, dark green from bile stasis and hardly granular at all. The external picture was typical of hypertrophic biliary cirrhosis. We were considerably surprised when we cut into it and found an immense tumor nodule occupying the central portion. It did not come to the surface anywhere or cause any localized deformity, and it would have been impossible to find at operation without actually cutting into the liver substance for at least a centimeter. The location was at first rather puzzling to make out. The common bile duct branched practically as it entered the liver. There was no external hepatic duct on either side. In the left lobe of the liver was a huge tumor mass. There was no tumor in the right half. The tumor had grown down the left hepatic duct and presented just at the beginning of the common bile duct, so that a little projecting nose on the end of the tumor was just big enough to occlude the right hepatic duct as well as the left one, so producing pretty complete obstruction on both sides. The cirrhosis in both lobes was about equal in amount. In spite of the high degree of obstruction he evidently must have been getting some bile through, because the feces were not clay colored and there was a considerable amount of bile pigment in the gall bladder. The bile there was green in color. At this stage of the examination we made our second diagnosis. We thought at first it was hypertrophic cirrhosis, as the surgeon naturally had, and then we thought that Dr. Jackson was right and we had a primary carcinoma of the liver. A few minutes later we found a good-sized nodule in the stomach which was obviously the primary carcinoma and this liver lesion was evidently a metastasis. The histology of the two is essentially similar, so that I think we can pretty safely rule out primary carcinoma of the liver. It is certainly not a hepatoma.

Have you any other comment, Dr. Leland?

DR. LELAND: No. I might say that we found nothing in the head of the pancreas and no dilatation of the common bile duct. We did very careful exploration of the large bowel and thought we did of the stomach. We thought the stomach had been pretty well ruled out. I am interested to know where the location of that tumor was, Dr. Mallory.

DR. MALLORY: It was on the anterior surface, about three centimeters below the cardia, in other words pretty high up under the diaphragm. We missed it, too, until the later stages of the autopsy. Our first palpation of the organs did not reveal it at all. It was only after we had the stomach out that it became obvious.

DR. LELAND: How large was it?

DR. MALLORY: Four or five centimeters in diameter.

A PHYSICIAN: Was there any examination of the stools?

DR. LYONS: We tested the stools for guaiac. They were negative.

DR. MALLORY: The case is of some interest in connection with the group of cases of biliary cirrhosis about which Dr. Chester Jones and I talked to you at the medical staff meeting. We had six cases of biliary cirrhosis in which the diagnosis rested on the clinical picture and the findings at biopsy. In none of these cases had the surgeon on exploration found any obstruction to the extrahepatic bile ducts. Here is a similar case with a biliary cirrhosis, very careful exploration, and proof even at autopsy that that was correct, and yet we have real obstruction as the basis of the biliary cirrhosis. It makes us realize that short of a complete and careful autopsy we cannot ever say there may not be obstruction in the biliary tract. It may be entirely intrahepatic.

A PHYSICIAN: May I ask Dr. Holmes what proportion of cases of carcinoma of the stomach might be missed by x-ray?

DR. HOLMES: I cannot answer that accurately, but my impression is that it is a very small one.

DR. MALLORY: The difficulties of examination become very much greater when the tumor is high up. If it actually involves the cardia it is easier than if it is a centimeter or two below, I imagine.

DR. HOLMES: If it is large enough to deform or stiffen the fundus so that the shape of the stomach does not change when the patient breathes, that helps us. Giving a small barium meal to outline the gastric mucosa ought to demonstrate the smaller lesions. The mistakes made ought to be in interpreting the findings rather than in missing the lesion completely.

CASE 19382

THE ASCHHEIM-ZONDEK TEST IN A CASE OF PELVIC DISEASE

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. NEIL L. P. CRONE:* A forty-two year old white married Italian born housewife presented herself in the Emergency Ward of this hospital complaining of epigastric pain of two

*Senior interne on the West Medical service.

months' duration. The history is inadequate and probably unreliable. She spoke no English and was very sick, and her family, who acted as interpreters, spoke English very little better.

Six months before admission she had a spontaneous miscarriage of a two months' fetus and following that had flowing for a period of ten days. After this her periods were regular for four months; the next month she missed entirely, and the following month she had flowing for three days a little prior to entry. Two months before admission she had steady pain in the epigastrium, in the right upper quadrant and above the symphysis in the midline. At this time she had some dyspnea on exertion; no other symptoms. Two weeks before entry the pain became worse and she took to bed. At this time the pain was localized entirely in the lower epigastrium and right upper quadrant. She developed a white vaginal discharge and burning on urination. One week before entry she developed cough with the production daily of one teaspoonful of sputum with no odor and no blood. At this time she had pain in the right shoulder aggravated by the cough, and had difficulty in getting her breath. For the past six months her bowels had been constipated. Her bowel movements had been entirely normal so far as we could find out. For a week before admission she had had drenching sweats every night. For four days she had had no bowel movement and had vomited three times a day.

The past history is irrelevant except that she had previously had two miscarriages, this one being the third.

Physical examination showed a fairly well developed woman sitting up in bed coughing violently, with some cyanosis, some evidence of weight loss and considerable dehydration. The cheeks were flushed, the teeth ill kept. The pharynx was injected. The right chest showed signs of fluid up to the angle of the scapula, rising in the axilla. Both chests were filled with moist râles. In the right upper quadrant there was some spasm and marked tenderness; no masses. The liver was not felt. Pelvic examination showed the perineum relaxed, a lacerated cervix and some whitish discharge. The fundus was felt anteriorly and was thought to be of normal size and firmness. There was a small hard mass in the posterior culdesac 5 by 3 or 4 centimeters. It was very hard but quite smooth and felt as if it might be encapsulated.

She had a hemoglobin of 70 per cent, red cell count 4,500,000. The white cell count varied between 12,000 and 19,000. The smear was negative. The sputum showed many spirochetes but never any blood and was negative for tubercle bacilli. Tests on the stools were negative, including guaiac. The urine was negative except for a slight trace of albumin and a few white cells. A blood culture and a Hinton test were negative. A van den Bergh test was normal.

Three chest taps were done on the right side for removal of fluid. The specific gravity of the fluid varied from 1.010 to 1.018. There were 2,400, 800 and 3,000 cells, 80 to 90 per cent lymphocytes. The last tap was frankly bloody.

X-ray examination of the chest showed a diffuse infiltrative process along the lymphatics. The examination showed also a right hydrothorax.

Because of the history of miscarriages and irregular periods, an Aschheim-Zondek test was done and was positive.

The patient's course in the hospital was steadily downhill. On the seventh day of her stay she expired.

Clinical Discussion

Dr. Crone: The x-ray findings were said to be consistent with either metastatic malignancy to the lung or miliary tuberculosis.

Our diagnosis at discharge was metastatic malignancy of the lungs and questionable chorio-epithelioma.

Dr. Fuller Albright: There was not much question that we were dealing with a case of metastatic malignancy with metastases in the posterior culdesac. The only question was where the primary source was. We were led to doing the Aschheim-Zondek rabbit test for the simple reason that she had had a miscarriage several months earlier. It might be said that she had menstruated after it, a fact which was against chorio-epithelioma. We injected the rabbit on two successive days; then opened it and found that it was still definitely negative. Inasmuch as so many cases of metastatic malignancy show a positive prolan A test we kept on injecting it for three more days. The rabbit finally died. At autopsy the remaining ovary,—the other having been taken out at the first operation—showed hemorrhagic changes such as we get in a positive pregnancy test. That led us to suspect that the patient did have chorio-epithelioma. However, cases of malignancy do show a positive prolan A test. That is not a positive pregnancy test since the enlargement of the follicles does not go on to corpora lutea formation. It does not show a complete pregnancy test, in other words. When these ovaries were sectioned it was very clear that they showed absolutely no suggestion of corpora lutea. There were many large ripened follicles, one of which was hemorrhagic. There was no reason to believe, therefore, that she did have chorio-epithelioma. We did not know that because the sections had not come through before she died. If the test had been done with mice we should not have had any trouble at all. The case never would have been thought to be chorio-epithelioma.

I have Zondek's book here, which was published in 1931. He was working on this test

when I was in Berlin in 1928. He did a large number of tests, and his book gives a summary of all the cases. He found this so-called positive prolan A test in many cases of malignancy, especially genital malignancy. What it means we do not know. He found it positive in 81 per cent of all his genital carcinomas, that is carcinomas of the cervix, fundus and ovaries. In extragenital carcinomas in women he found it positive in 36 per cent of all cases.

DR. EARLE M. CHAPMAN: Those tests are positive only after repeated urinary injections. The pregnancy test requires only one injection.

DR. ALBRIGHT: We do not do the test as a rule with rabbits. We use mice for prolan A tests and rabbits for pregnancy tests.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Chorio-epithelioma of the uterus?
Metastatic malignancy.

ANATOMIC DIAGNOSES

Carcinoma of the stomach with metastases to the lungs and the mesenteric and retroperitoneal glands.
Carcinomatosis.
Hydrothorax, right.
Hydronephrosis, right.
Arteriosclerosis, slight.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: The autopsy here showed that we were again dealing with unsuspected primary carcinoma of the stomach, a distinctly larger one than in the last case, with a big ulcerated crater. It is rather surprising that the test for occult blood in the stools was negative. Metastases had extended primarily to the lungs and pleura. There were relatively few metastases within the abdomen, though some were found in the region of the tail of the pancreas.

The uterus showed a moderate degree of hyperplasia of the endometrium; otherwise the section was negative.

DR. ALBRIGHT: What did the ovaries show?

DR. MALLORY: One ovary was atrophic. The other had a few small old follicular cysts in it.

A PHYSICIAN: Was the pituitary considered normal?

DR. MALLORY: So far as we have been able to make out in the sections it was. There was nothing very striking about it.

A PHYSICIAN: Were there metastases in the pelvis?

DR. MALLORY: Yes, in the posterior culdesac. That is the commonest site of metastases for an intra-abdominal tumor.

A PHYSICIAN: Do any tumors except the chorio-epitheliomas ever give the full test that is typical of pregnancy?

DR. ALBRIGHT: There have been one or two sporadic ones reported. I have not yet followed the New York reports very closely. I think they have had a few others.

A PHYSICIAN: Has any work been done to show that it is commoner with chorio-epithelioma than with sarcoma, for instance?

DR. ALBRIGHT: There are no sarcomas mentioned in Zondek's series. The tests are all negative in fibroids and other benign tumors.

DR. GEORGE W. HOLMES: The work in New York is on teratomas of the testicle, is it not?

DR. ALBRIGHT: Chorio-epitheliomas of the testicle are teratomas in the strict sense, I believe.

DR. MALLORY: Although a very small proportion of them show a chorio-epitheliomatous appearance histologically.

DR. HOLMES: Do I understand that this test would be negative if there were no tumor present?

DR. ALBRIGHT: Zondek says here that any carcinoma of the cervix ought to give it in practically every case. If the carcinoma of the cervix is removed, the test becomes negative. If any part of the tumor is left, the chest continues to be positive.

DR. HOLMES: What about the patient who has had a carcinoma removed?

DR. ALBRIGHT: I think that in carcinoma of the cervix it would be very satisfactory to know it was negative after removal.

DR. HOLMES: If the question is raised in any case and all the examinations are negative, could we use that test to prove that the patient did not have carcinoma?

DR. ALBRIGHT: We have just learned that the test is positive in 81 per cent of the cases of genital carcinoma.

We have really not done very many of these tests in this hospital, only a few here and there.

The New England
Journal of Medicine

SUCCESSOR TO
THE BOSTON MEDICAL AND SURGICAL JOURNAL

Established in 1828

Published by THE MASSACHUSETTS MEDICAL SOCIETY under the jurisdiction of the

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SUBSCRIPTION TERMS: *$6.00 per year in advance, postage paid for the United States, Canada $7.04 per year, $8.52 per year for all foreign countries belonging to the Postal Union.*

Material for early publication should be received not later than noon on Saturday. Orders for reprints must be sent to the Journal office, 8 The Fenway.

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Communications should be addressed to The New England Journal of Medicine, 8 The Fenway, Boston, Mass.

THE CARE OF PERSONS "ON RELIEF"

HARRY L. HOPKINS, Federal Emergency Relief Administration, has released for general information some of the rules governing the expenditure of Federal funds for medical, nursing and dental care for destitute unemployed ill persons.

Federal relief money for medical care under this plan is available for office and home treatment by physicians, but not for hospitalization or service in nursing homes or clinics. Office treatment of ambulatory patients shall not supplant the service of existing clinics in communities.

Agreements between relief officials and physicians provide for treatment within two weeks, limited to two visits for acute illnesses. If the service extends over two or three months, only one visit a week will be approved except after reinvestigation.

For obstetric cases the allowance to the doctor is fifteen dollars which includes ante- and postpartum care. A certain elasticity in the rules provides for special consideration in cases out of the ordinary. State and local organizations will be asked to designate advisory committees for the purpose of elaborating adequate policies.

While this plan will be of distinct help to physicians who are now extending charity to indigent persons who are suitable for Federal relief, one does not have to be devoid of imagination to expect that the operation of these rules will bring to bear on doctors more or less constant adjustment to the findings of the committees in charge of the rules which may be as difficult or even more so than those found in dealing with the workmen's compensation proceedings. Much will depend on the committees in charge as well as the disposition of doctors to regard the necessities of the patient as overshadowing a niggardly or selfish attitude toward specific cases. If the officials are inclined to dictate the details of medical care, there will arise in the mind of the doctor a feeling of opposition to and distrust of the plan. To some the bogey of state medicine will appear, but, if both parties in interest will appreciate the opportunity to relieve human suffering with fairness in every particular, great good will result. It has long been recognized that the burden of medical care of the indigent should not be carried by the doctor but that it is a community problem as much as that of food and other necessities.

We look forward to a more general recognition of community responsibility for the medical care of the indigent.

THE DEAF IN THE UNITED STATES

TEN million Americans are deafened and few of the remainder have perfect hearing according to statements by the Mandeville Press Bureau of New York City. The normal ear records sound vibrations of 30 to 30,000 per second and those with unusually acute hearing may recognize 35,000 per second.

If the auditory nerve is not impaired, sound may be transmitted through the bony structures by specially contrived agencies. The range of hearing may often be increased through practice and, since it is estimated that there are 3,000,000 school children in this country with subnormal hearing, there is a great responsibility on parents and physicians relating to the problems of deafness. Many of the causes of deafness are preventable because ear troubles are often the result of infections advancing through the Eustachian tubes. The importance of so-called colds in relation to deafness is not generally recognized and, until there is more general recourse to medical care and prevention,

much reduction of deafness cannot be expected. These three million children have a heavy handicap in the competitive conditions of modern life.

THE PROBLEMS INCIDENT TO BLINDNESS

THE problems incident to blindness in Massachusetts appear in the Annual Report of the Division of the Blind for the year ending Nov. 30, 1932, in the service to 3049 blind persons with 4,520 of these afflicted persons on the register, classified as follows:

Under 5 years	46
Between 6 and 20 years	548
Between 21 and 50 years	1,302
Between 51 and 70 years	1,459
Over 70 years	995
Unknown	170

Four hundred and forty-two new cases were registered during the year including 149 boys and 132 girls most of whom were between the ages of six and ten. There are thirty-seven sight-saving classes in larger centers throughout the State and several workshops where chairs are reseated and brooms and rugs are made.

Financial assistance was given to 965 adults amounting to $174,433.43. This is probably an illustration of methods employed in other states and places before the people an illustration of the importance of the demand for help. Here as in most charitable endeavors the financial problem is serious.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MILLER, RICHARD H. A.B., M.D. Harvard University Medical School 1910. F.A.C.S. Associate Visiting Surgeon, Massachusetts General Hospital. Assistant Professor of Surgery, Harvard Medical School. Address: 264 Beacon Street, Boston. Associated with him is:

BARTLETT, MARSHALL K. B.A., M.D. Harvard University Medical School 1928. Assistant in Surgery, Massachusetts General Hospital. Their subject is "Umbilical Hernia." Page 565.

MOORHEAD, JOHN J. B.S., D.S.M., M.D. New York University Medical College 1897, F.A.C.S. Professor of Surgery Post Graduate Medical School, Columbia University. Director of Department of Traumatic Surgery Post Graduate Hospital. Surgical Director of the Reconstruction Hospital Unit. His subject is "Relation of Trauma to Hernia." Page 568. Address: 115 East 64th Street, New York City.

MARBLE, HENRY C. A.B., M.D. Harvard University Medical School 1910. F.A.C.S. Surgeon to Outpatients, Massachusetts General Hospital. Surgeon to the Chelsea Memorial Hospital and to the United States Veterans' Hospital, Bedford, Mass. Consulting Surgeon, Faulkner Hospital. His subject is "Recurrent Herniae." Page 574. Address: 270 Commonwealth Avenue, Boston.

HOLLAND, JAMES H. New England District Claims Manager of the Liberty Mutual Insurance Company, Boston. His subject is "Hernia From the Compensation Insurance Standpoint." Page 579. Address: Park Square Building, Boston.

BOWLER, JOHN P. A.B., M.Sc., M.D. Harvard University Medical School 1919. F.A.C.S. Dean, Dartmouth Medical School. Surgeon, Mary Hitchcock Memorial Hospital. His subject is "Medical Care of the County Poor." Page 586. Address: Hitchcock Clinic, Hanover, New Hampshire.

MILLER, RALPH E. B.S., M.S., M.D. Harvard University Medical School 1928. Assistant Professor of Pathology, Dartmouth Medical School. His subject is "The Secondary Nodules of Lymphatic Tissue. Their Relation to Immunity." Page 591. Address: 9 Downing Road, Hanover, New Hampshire.

MATZ, PHILIP B. M.D. For further information see This Week's Issue, page 560, issue of September 14, 1933.

MISCELLANY

WHERE THE TWO-YEAR MEDICAL STUDENTS OF DARTMOUTH COLLEGE ARE TAKING ADVANCED COURSES

Although the Alumni of the Dartmouth College Medical School have from time to time advocated extending the two years of study in medicine to a full four year course, there are no indications of this change at present.

It is of some interest to note the selection of medical schools for the other two years by these students. Of those who have completed the two years at Dartmouth, McGill now has eleven, Harvard seven, Pennsylvania and Rush five each, Columbia four, Johns Hopkins, Cornell, and Washington University two each, Jefferson, Colorado, Western Reserve, Stanford and Bellevue one each.

WHAT IS NEW IN TUBERCULOSIS PATHOLOGY

Sir Humphry Rolleston, who reviewed the subject of non-pulmonary tuberculosis, dwelt especially on chronic hyperplastic tuberculosis of the

intestines and the accompanying hyperplastic peritonitis. He made the interesting suggestion, proposed by preceding investigators, that these unusual forms of tuberculosis, in which tubercle bacilli are difficult to find, might be due to different phases of the several stages of the life cycle of the bacillus of tuberculosis, recently described by several investigators.

Drs. Petroff and Winn of Trudeau, New York, reported further developments in their study of colony variation in the growth of avian tubercle bacilli. From a single strain of this bacillus they separated out four types, the extremes of which formed "smooth" and "rough" colonies on culture media and were respectively virulent and avirulent for chickens.

A paper presented by Dr. Daines of Salt Lake City may explain some of the positive tuberculin reactions reported from time to time leading to slaughter of cattle subsequently proved not to be tuberculous. He described acid-fast bacteria, apparently not tubercle bacilli, obtained from certain skin lesions of cattle reacting positively to tuberculin, but not tuberculous, as shown by postmortem examination. The bacteria, which appeared to be a previously undescribed variety, and the skin lesions they caused, evidently induced a sensitization to tuberculin.

A complicated life cycle of the tubercle bacillus, in which several granular phases occur, was reported by Dr. Kahn of Cornell University. He embedded single colonies of tubercle bacilli growing on solid culture media and sectioned them. Stained by the usual methods for tubercle bacilli, sections showed zones of acid-fast, partially acid-fast and non-acid-fast bacteria, conforming closely to the types previously described by Dr. Kahn in his cultures from single organisms.

At a later meeting Dr. Wyckoff of the Rockefeller Institute, New York, also presented a paper on the growth of tubercle bacilli, which was apparently not in agreement with Dr. Kahn's work. Dr. Wyckoff used the ingenious method of motion photography of the growing bacteria on clear solid media. Several kinds of tubercle bacilli, chiefly derived from tuberculosis of cold-blooded animals, reproduced by the simplest of all methods, elongation and fission into two new organisms like the original.

Another paper, presented by Drs. Soper, Alpert and Adams, dealt with the relative immunizing properties of a "smooth" type variant of bovine tubercle bacilli, killed by heat, the well known living attenuated BCG strain of bovine bacilli, and living human type bacilli. The heat-killed bacilli caused the greatest protection. Dr. Willis of Detroit described a surprising frequency of tubercle bacilli in the urine, as well as sputum, of children with childhood type tuberculosis.

The second symposium was devoted to silicosis. (A forthcoming number of *Abstracts* will be devoted to this subject.)

PROGRESS IN RESEARCH

A third symposium was given to reports from investigators working under grant from the National Tuberculosis Association. Dr. Richardson of Cornell University reported experiments by himself and colleagues on the respiration of tubercle and related bacilli. The most significant fact was the high sensitivity of the pathogenic acid-fast bacteria to deprivation of oxygen, a characteristic probably playing a rôle in the limitation of their growth in the animal body.

Several papers dealt with the proteins of tubercle and related acid-fast bacilli. It was brought out clearly by several investigators that the pure proteins of these bacteria are capable of sensitizing the animal body so that subsequent injections of the same material into the skin elicit an inflammatory response much like that caused by tuberculin in the tuberculous animal. Dr. Aronson of the Phipps Institute reported that while the protein MA100 almost exactly paralleled Old Tuberculin as skin-testing material, it possessed the capacity, on repeated injections in children, of sensitizing the skin so that false positive reactions were given with later higher dosages.

Dr. Sabin and colleagues of the Rockefeller Institute reported results of similar import from animal experimentation. Their results on inoculation of animals sensitized to one of the proteins of the tubercle bacillus also brought out clearly that the sensitization conferred no increased resistance to infection.

Dr. Seibert of the Phipps Institute reported experiments indicating that the sensitizing character was referable to the high molecular weight of the proteins, and showed that by two methods it was possible to reduce the molecular weight and prepare pure protein substances with full skin-testing potentiality and accuracy, but virtually devoid of sensitizing character. The first consisted in boiling one of the purified proteins in weak alkali (pH 9.0-10.0) and again isolating the protein. The second in isolating the protein directly from an Old Tuberculin prepared by the usual heating method, but derived from a culture on a non-protein synthetic medium instead of the usual glycerol-peptone-meat infusion broth.

From the discussion following, which was opened by Dr. Long, it seemed clear that the preparation finally to be recommended to replace the O.T. of common use, was a protein of non-sensitizing character but full potency isolated by one of these methods, and Dr. Long reported that a plan was under way to make an objection-free preparation available shortly.

Two papers were presented by Drs. Mariette, Fenger, Duncan and others from the Glen Lake Sanatorium, which showed a surprising lack of specificity in both the pure lipoids and pure proteins of the acid-fast bacilli when used respectively in serological and skin tests. Purified protein pre-

pared by the method for MA100, but from timothy instead of tubercle bacilli, appeared even more potent than MA100 in eliciting skin reactions in tuberculous subjects.

At the final session Dr. James Alexander Miller of New York called attention to the newly recognized frequency of hematogenous as well as bronchogenic spread in pulmonary tuberculosis.

Drs. Wiseman and Doan of Ohio State University reported progress in their work on blood changes in tuberculosis, showing that not only do important changes in the number of lymphocytes occur, but that qualitative variations, referable to age, occur in the lymphocytes, which are of considerable prognostic significance.

Drs. Schroder and Park of New York compared the effect of dead tubercle bacilli and living BCG in inducing tuberculin sensitiveness in children. Positive Mantoux tests occurred in almost all of the children receiving BCG, and in only about a third of those getting dead bacilli, and sensitiveness lasted longer in the former group.

Dr. Burke of Ray Brook, New York, showed that it was possible by intratracheal injection of tubercle bacilli in normal tuberculous rabbits to produce forms of tuberculosis roentgenologically and pathologically comparable to childhood and adult tuberculosis in man, a result of obvious value for studies on the pathogenesis of the two types of the disease.

Drs. Johnston, Howard, and Maroney of Detroit gave some long-desired data on the development and course of the tuberculin reaction following first infection in childhood. Frequent tuberculin testing and x-raying of a group of children showed that the progress of healing childhood infection from primary lung infiltration to calcification of the corresponding hilum lymph nodes, averaged about two years, during which the tuberculin reaction became positive, reached a maximum of intensity, and then waned to a lower level.

An experimental study by Dr. de Savitsch and colleagues from the University of Chicago on the effects of a combination of irradiated ergosterol or parathormone with tuberculin in tuberculous animals, not only confirmed previous results on the calcifying action of the first two substances for tubercles, but indicated also that it may be possible to enhance this effect by increasing the blood supply around tubercles by the focal reaction following injection of tuberculin. — *Brief abstracts of the papers and symposiums of the Pathological Section of Meeting of the National Tuberculosis Association, June 1933.*

PSYCHIATRY AND SOCIAL SCIENCE

Two important educational sections of interest to physicians are provided in the University Extension classes as set forth in the *Bulletin of the Massachusetts Department of Education.* One is the lectures by Henry B. Elkind, M.D., on Mental Hygiene and Psychology, Applied Psychology by Irving C. Whittemore, and Psychology of Personality by A. A. Roback; the other on Society Problems by Dr. Douglas A. Thom and David Davies Vaughan.

Physicians should be interested in better understanding of these subjects by the laity and hence suggest enrollment in these classes to those who have time and can afford the expense.

More general understanding of these subjects will develop, in the public mind, the importance of applied psychology and social science.

ENCEPHALITIS IN ST. LOUIS

Late reports are that the outbreak of encephalitis in St. Louis County seems to have reached its peak with 144 deaths out of about 800 cases. Stringent regulations of funerals for victims of the disease are in force on the assumption that friends of those who have died may be carriers. A small epidemic exists in Independence, Mo.

THE AWARD OF THE WILLARD GIBBS MEDAL

On September 13 at a meeting in Chicago the Willard Gibbs Medal was awarded to Professor Richard Willstaetter of Munich. The occasion was the central event of the program of the Century of Progress meeting of the American Chemical Society attended by prominent chemists of this and foreign countries. This medal is awarded annually and is regarded as one of the highest honors which is bestowed on eminent scientists by the Society.

Professor Willstaetter is a Nobel Prize winner in chemistry and in an interview, as reported in the *New York Times*, is quoted as believing that the mystery of cancer and of life lies in recently discovered enzymes and that life becomes more understandable in terms of pure chemistry.

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

ARTERIOSCLEROSIS*

BY PAUL D. WHITE, M.D.

Arteriosclerosis literally means hardening of the arteries, but this is only the end stage of a process of softening or weakening of the inner and middle arterial coats which may go on for many years before the hard lime salts are deposited. The condition is universal and the process of deterioration begins in youth.

Arteriosclerosis is not strictly abnormal if we think of it as a wearing-out process in very old persons, although in future centuries even this occurrence may be regarded as pathological if it be-

*May 16, 1933, Station WBZ, 4:30 P.M.

gins within the first hundred years of life. According to our present ideas arteriosclerosis takes on serious significance if it causes important symptoms or signs of disability or death before the age of seventy years, and the earlier the age the greater the significance. Much is said about it nowadays and there is a general impression that it occurs more often at earlier ages than it used to do. This impression may be justified by facts, but we must consider two possible explanations other than that fewer persons escape the process; these explanations are first, that more persons now survive infancy with its illnesses which were once formidable but now are largely under control, some of these survivors having inherited poor arterial tissue, and secondly, that the diagnosis of "hardening of the arteries" is more easily, accurately, and often made nowadays than formerly. Be that as it may, it does appear true that our fathers did not live so long as did our grandfathers, and that we are not living so long as did our fathers. The much vaunted increase in the duration of human life in our time is due wholly to the survival of many individuals through childhood and early adult life who would once have died of dysentery in infancy or of typhoid fever or some other such infectious disease afterwards. None of this increase in the duration of life is due to an increase of the life span itself. It is this problem that now demands our urgent attention. We must do something to prevent the appalling waste of life at its zenith; this waste of life is largely the direct result of arteriosclerosis.

Hardening of the arteries is not a modern disease; it existed in ancient days as evidenced by finding it in Egyptian mummies, but it was clearly recognized as a disease only in relatively modern times, that is within the past 200 years. However, that excessive nervous strain and faulty diet were causes of disability and death was known to the medical faculty of the University of Salerno in Southern Italy in the Middle Ages, even though the mechanism by which such disability was actually produced was not known. In an early English translation in 1541 of the "Regiment of Helthe" of Salerno one finds the following observations: "He that desireth helth of body must eschew and avoyde great charges, thoughts, and cares. . . . The second doctrine is to eschewe anger. . . . . The thyrde doctryne is to eate and drynke soberly. . . . The fourth doctrine is to make a lyght souper. . . . The fifth doctrine is to walke after meate. . . . The sixth doctrine is to eschewe slepe immediately after meate. . . . Finally the auctor sayth, that who so wyl observe the forsayde doctrines, shall lyve longe in good helthe and prosperite."

Arteriosclerosis is widespread throughout the body and throughout the world, affecting all arteries and all races, although to different degrees. It attacks all kinds of animals, too, especially the more active mammals. It occurs in man at all ages but naturally increasingly with advancing years. Both sexes are affected, but the male sex preponderantly at earlier ages, apparently because of the greater strain to which males are as a rule subjected.

The cause of arteriosclerosis is not clearly understood in most instances. Through "wear and tear" at points of unusual pressure, stretching, or bending, the wall weakens. Fat is deposited as "atheroma," which literally means pudding, in spots in the intima or inner wall, or the muscle of the media or middle coat degenerates. In the course of time lime salts are deposited in these damaged spots and the artery becomes stiff and narrowed. The chemistry of all this change is still obscure but certain factors are known which favor the onset of this change; these are excessive work, excessive blood pressure, diabetes, lead poisoning, syphilis in the case of certain arteries and inherited tendency. Other factors have also been blamed but they are open to question—they are excessive food, certain foods like protein taken to excess, tobacco, and infectious disease in general. Some day we shall have answers to these questions—at present they remain largely unsolved.

What are the symptoms and signs of arteriosclerosis? How does it show itself? Mostly through disturbance of function of whatever tissue or organ is affected by the decrease of blood supply resulting from the narrowing of the arteries. Of course in some parts of the body as at the wrist or on the foot or on the forehead it is possible to feel the arteries and to judge how hard they are directly, but most of the important arteries of the body are internal and so not to be reached except sometimes by x-ray study or by special tests or by observations as to the function of the organs supplied by them.

Thus it is obvious that the symptoms and signs of arteriosclerosis are legion and extend from the crown of the head to the soles of the feet. I shall now name instances of the more important or more obvious results of localized arteriosclerosis. Beginning with the head these are whitening of the hair due to local changes in the arteries supplying the hair follicles, loss of elasticity of the skin, apoplexy and various other disturbances of the brain due to hardening of the arteries there, together with high blood pressure, cataracts in the eye from opacities of the lenses, and deafness from decrease in the blood supply to the inner and middle ears. In the thorax we find elongation and stiffening of the great artery called the aorta, angina pectoris and coronary thrombosis due to arteriosclerosis of the important little coronary arteries that supply the heart muscle with blood, and changes in the arteries of the lungs following extensive lung diseases and increased blood pressure in the lungs. Next there is Bright's disease from affection of the small arteries of the kidneys. Finally in the extremities reference should be made to cramps in the muscles on exercise (called inter-

mittent claudication) caused by sclerosis of the arteries going to the muscles, and finally to atrophy and even gangrene of toes and fingers from marked changes in the arteries supplying the extremities. Frequently several organs may be affected at the same time but often only one is damaged to any notable degree. The most serious effects are on the heart and brain; of next importance is limitation of the blood supply to the kidneys.

The treatment of arteriosclerosis affords little that is curative. Alleviation is, however, often possible through various measures, particularly that of reducing the amount of work thrown upon the affected organ, as in the case of heart or kidneys. There are no magical drugs or remedies although from time immemorial such drugs or remedies have been advised. The fountain of youth has not yet been discovered. We must neither foster unwarranted hopes nor yet should we consider that nothing can be done when arteriosclerosis causes trouble. Frequently symptoms can be relieved and life prolonged for years by common sense measures known to all physicians.

Of far greater importance than the treatment of arteriosclerosis is, of course, its prevention. Since we know some of the factors behind it, we can accomplish definite results in this very vital phase of preventive medicine. The two important points in this direction are first, the establishment of good habits of health in early life—exercise, rest, relaxation, diet, all in moderation—and secondly, the avoidance of infections and poisons (which may or may not include tobacco—no one knows for certain). These are very difficult measures and at present are impossible to apply in full, but the more complete their fulfillment the more delayed the appearance of arteriosclerosis, always bearing in mind, however, that a large share of responsibility appears to be that of inheritance.

Finally, many individuals, perhaps justifiably at times, will not wish to carry out all the measures that may prevent their being early crippled or killed by arteriosclerosis. A carefully planned existence with a view only to health may be too humdrum. Last year in a radio message Dr. Harold Stevens quoted a verse of Edna St. Vincent Millay which well expresses this idea:

"My candle burns at both ends;
It will not last the night;
But Ah, my Foes and Oh, my Friends—
It gives a lovely light!"

In the final analysis, however, the maximum of happiness and usefulness can be secured, as a rule, by heeding the simple sensible tenets of healthy living, thereby avoiding early disability and death with all the burden and misery that such trouble brings upon family, friends, and community.

CORRESPONDENCE

PRELIMINARY REPORT RESPECTING POSTGRADUATE INSTRUCTION

The Massachusetts Medical Society
8 The Fenway, Boston, Mass.

September 15, 1933.

Editor, The *New England Journal of Medicine*,

The Executive Committee wish to give you the following information in regard to the response of the doctors to the Committee's program on Postgraduate Instruction.

District	Membership (Active)	Cards Received
Barnstable	37	8
Berkshire	109	28
Bristol North	64	18
Bristol South	199	41
Essex North	192	39
Essex South	253	49
Franklin	32	11
Hampden	291	47
Hampshire	55	19
Middlesex East	96	16
Middlesex North	121	31
Middlesex South	768	109
Norfolk	715	81
Norfolk South	115	22
Plymouth	129	34
Suffolk	738	66
Worcester	381	74
Worcester North	86	15
Total	4381	708

We will keep you informed from time to time in regard to further data as they are received.

Very truly yours,
LEROY E. PARKINS, M.D., *Secretary*,
Executive Committee on
Postgraduate Instruction.

MAGGOTS

Editor, *New England Journal of Medicine*,

Because the much talked of maggots still seem to crop up as a live issue; because there seems to be much misunderstanding as to their value; because there seem to be no definite data as to results from their employment, the time seems proper for a line about them.

Moreover, I feel a certain responsibility for inviting them here to Boston.

In the Spring of 1930, I had the privilege of visiting the late Dr. Baer's admirable fly-farm, and seeing his cases, fresh and cured, at the Painter Home and in the Marburg Building at Johns Hopkins with him.

Shortly thereafter we arranged to send two young research men from the Boston City Hospital to Baltimore to learn the technique, by courtesy of Dr. Baer, and on their return the raising of maggots was begun and very successfully carried on to date.

This was early in the summer of 1930.

From then on, cases have been treated in some considerable number, and it is hoped that some official data as to results may be forthcoming, soon.

Meanwhile I am rather constantly asked for an opinion as to the method.

A statement may not come amiss.

Not a few of the cases, earlier, were mine, and beyond this I have been interested to check results on these and on other cases, as I could.

What I can give is my own opinion and conclusions, only.

It still seems to me that the repair in accessible osteomyelitic wounds and the like is somewhat more prompt, that defects fill with granulations more quickly, that the massive scars of repair are fuller and softer under maggot treatment. In this class of cases the end results as to cures also are as good as we get in expert hands by other methods, perhaps better.

In cases with deep wounds—the sinus cases—so unsatisfactory by any method we have used in the past (the cases I hoped the maggots would clear up for us), the maggots seem entirely useless. Despite every artifice of driving them in with strong light, etc., they are not deep feeders. So, for these cases, they go to sit with the tubes of bismuth paste and other hopes of former days.

I understand Baer, later, did not accept cases of this class, nor did I see any such in his wards.

One function of the maggots is worth more study. In accessible wounds with sloughing tissues and sluggish granulations they seem to clean the field better than the digestant fluids (like enzymol) and probably encourage granulations.

In cleaning up pressure sloughs (as bedsores, etc.), I have seen nice results, and was much impressed with Baer's demonstration of the clean-up of a slough by tubercular costochondral ulcer, often operated.

To sum up, there is no real argument save the dollar argument against using maggots, but there seem to be definite bounds to their field of real effectiveness.

To urge their use beyond such bounds is perhaps not quite scientific, or frank.

FREDERIC JAY COTTON.

REORGANIZE THE HOSPITALS AND HELP THE PHYSICIANS

The physician is the one who is called upon at all times regardless of the time, weather, or where he may be to answer the call of the sick and when he performs his duty with the best that is in him and it comes time to pay, the usual answer is: "I will pay you to tomorrow, doctor," and sometimes he doesn't even get a promise to pay, but only "thank you". Many of those promises to pay are never remembered.

Editorials and pictures have been printed in various newspapers and magazines showing how quickly the people want us to come when they are sick, but when it comes to paying the physician's bill, the answer is, "The doctor's bill! Oh, well, he can wait." Just suppose that doctors responded to calls with the same speed that most patients show in paying their bills. I hate to state what would happen to those sick patients.

I am glad to be able to thank M. S. Barnard from Marblehead Neck for the splendid letter which he wrote to the *Herald* and which was printed in the morning *Herald* of September 1, 1933, under the heading of "Doctor Is Forgotten Man When the Bills Are Paid." More power to his pen. We need good souls like him and hope that all who read his letter in the *Herald* will feel the same way.

We hear the cries of the masses that millions are out of work. Do you know that we have about 160,000 physicians in the U. S. A. and that many of these physicians who give everything that they have such as money, health and their best years, finally find that they are worse off than the ordinary laymen? We also hear complaints that people work too many hours. Has it ever occurred to you that the physician is the worst sweat shop worker of all? Why should a physician have such unusual problems? We are always willing to give charity to all who need our services, donate to all worthy causes, give our services gratis to hospitals and out-patients and what do we get in return? Nothing. As I have already expressed my opinions on former occasions, physicians are the most charitable of any class. But when we learn that a large majority of the patients who get free services at the hospitals and clinics can afford to pay for services, it is about time that something was done to such patients.

Is there any earthly reason why physicians should be forced to take some of their patients to court in order to collect fees or professional services? And what those patients do say in court or elsewhere when legal action is brought against them! I feel that the medical profession should never be forced to take such measures, but am sorry to state that in many instances unless a court action is brought the physician would never be paid.

We must reorganize charity or free services. Every case which applies for free services must be thoroughly investigated and a penalty imposed by law for those who take such free service and can pay. Every out-patient department should have a sign conspicuously placed and printed with large letters, saying: "This is a charity clinic." We must arouse the Board of Trustees of hospitals to the fact that the main object of the out-patient

department of hospitals should not be to show that they treat thousands of patients in the hospital proper and out-patient with a big deficit, but that they give services to all who need charity and so, by eliminating those who can pay for services, greatly reduce the expenses of the hospitals. Awake, Boards of Trustees, reorganize your hospitals, and by so doing you will also give a helping hand to the general practitioner and the medical profession as a whole.

BERNARD ZUCKERMAN, M.D.
1011A Blue Hill Avenue,
Dorchester, Mass.

RECENT DEATH

BUSH—ARTHUR DERMONT BUSH, M.D., of Decatur, Georgia, died September 6, 1933 at the home of a relative in Albany, N. Y., after an illness of thirteen years.

Dr. Bush was born August 18, 1875. He graduated in medicine from the Atlanta College of Physicians and Surgeons in 1901 and was a practicing physician in Decatur, Georgia, and a teacher for twenty years. His publications include four texts and various research problems. He joined the Massachusetts Medical Society in 1909 and was also a Fellow of the American Medical Association. He was a Mason, a member of the A.A.A.S., and the Sigma Xi.

NOTICE

CLINICS AT THE PETER BENT BRIGHAM HOSPITAL

On Thursday, October 5, in the amphitheatre of the Peter Bent Brigham Hospital at 3:30 P.M. there will be a clinic conducted by Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, to which physicians are cordially invited. These clinics will be repeated weekly on Thursdays at this hour until May.

REPORT AND NOTICE OF MEETING

FRANKLIN DISTRICT MEDICAL SOCIETY

The September meeting of the Franklin District Medical Society was held at the Weldon Hotel, Greenfield, at 11 A.M., September 12.

The society was extremely fortunate in its program, for Dr. H. M. Clute of the Lahey Clinic presented a most interesting and clean-cut paper on Obstructive Jaundice—Its Diagnosis and Treatment. He illustrated his talk with lantern slides.

Dr. Robey, newly elected President of the Massachusetts Medical Society, with Dr. T. J. O'Brien, were also present. Dr. Robey spoke on the postgraduate work to be started this fall and roused such enthusiasm that nearly every member present expressed his desire to participate in the course.

Dr. O'Brien was very earnest in presenting the present standing of medicine and decried the lack of coöperation and apathy of the average doctor especially as regards legislative matters. The consensus seemed to be that this was one of the best meetings in a long time. Twenty-five members were present out of a membership of thirty-two.

CHARLES MOLINE, *Secretary.*

THE ANNUAL CONFERENCE OF SECRETARIES OF THE STATE MEDICAL ASSOCIATIONS

This conference takes place Friday, September 22, and continues through the forenoon of Saturday, September 23. Editors of the State Medical Journals and Presidents of State Medical Societies are also invited. Dr. T. J. O'Brien, representing Dr. Robey, President of the Massachusetts Medical Society, will attend.

The subjects discussed are the Quality of Medical Care by Dr. Dean Lewis, President of the American Medical Association; the Michigan Survey by Dr. F. C. Warnshusis; Social Dangers of Oversupply of Physicians by Dr. Walter L. Bierring, President-Elect of the American Medical Association; Work of the Educational Committee of the Illinois Medical Society, by Dr. H. M. Camp; Postgraduate Work, by Dr. Thomas P. Farnum; Medical Defense, by Dr. J. E. Tuckerman; Legislative Problems, Federal and State, by Dr. W. C. Woodward, Director of the Bureau of Legal Medicine and Legislation, American Medical Association; and Work of the Committee on Ethics of the Connecticut State Medical Society, Dr. C. W. Comfort.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

September 22 and 23—Annual Conference of Secretaries of the State Medical Associations. See notice above.

September 25-30—The American Congress of Radiology will be held at the Palmer House, Chicago. For particulars write to Dr. Benjamin H. Orndoff, 2561 North Clark Street, Chicago.

September 29 and 30—New England Surgical Society at Boston.

October 5—Clinic at the Peter Bent Brigham Hospital. See notice elsewhere on this page.

October 9-12—The American Public Health Association will hold its sixty-second annual meeting at Indianapolis. For details on the scientific program write to the American Public Health Association, 450 Seventh Avenue, New York, N. Y.

October 9-13—The Clinical Congress of the American College of Surgeons will hold its twenty-third annual session at Chicago. Headquarters for the Congress will be at the Stevens Hotel.

October 13, 1933 - May 11, 1934—William Harvey Society. (Eight Lectures. Monthly.)

October 16, 17, 18—Massachusetts State Nurses' Association, Hotel Kimball, Springfield, Mass. For details write Helene G. Lee, R.N., 420 Boylston Street, Boston.

October 18—New England Physical Therapy Society. First Fall Meeting. Detailed announcement later. Dr. Arthur H. Ring, Secretary, 163 Hillside Avenue, Arlington, Mass.

October 23 - November 3—The 1933 Graduate Fortnight of the New York Academy of Medicine. The theme during the two weeks of intensive study will be Metabolic Disorders. A complete program may be obtained by writing Dr. Frederick P. Reynolds, The New York Academy of Medicine, 2 East 103rd Street, New York City.

October 25-30—International Congress on the Scientific and Social Contest Against Cancer will be held at Madrid. For information address Dr. Julio Bejarano, Atocha, 104. Madrid, Spain.

October 28—The American Board of Dermatology and Syphilology will hold written examinations for candidates in the following cities: New York City; St. Louis, Mo.; Boston, Mass.; Cleveland, Ohio; San Francisco, Calif.; Philadelphia, Pa.; Chicago, Ill. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

December 15 and 16—The American Board of Dermatology and Syphilology will hold oral examinations in New York City. Information may be obtained from the Secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

April 16-20, 1934—The American College of Physicians will hold its Eighteenth Annual Clinical Session in Chicago at the Palmer House. For information write Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

July 24-31, 1934—The IVth International Congress of Radiology will be held in Zurich under the presidency of Professor H. R. Schnitz. General Secretary Dr. H. E. Walther, Gloriastrasse 14, Zurich.

DISTRICT MEDICAL SOCIETY

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of November, January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, Secretary.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

Schedule 1933-1934

Meetings will take place on November 8 at the Bear Hill Club, Stoneham; January 10 at Melrose; March (2nd Wednesday) at Wakefield, and May (2nd Wednesday) at Winchester.

ALLAN R. CUNNINGHAM, Secretary.
76 Church Street, Winchester, Mass.

MIDDLESEX NORTH DISTRICT MEDICAL SOCIETY

Meetings will be held on October 25, January 31, and April 25 (Annual Meeting).

T. A. STAMAS, Secretary.

NORFOLK SOUTH DISTRICT MEDICAL SOCIETY

October 5—12 noon at Norfolk County Hospital. Speaker: Dr. L. P. Crawford. Subject: Acute Intestinal Intoxication.

November 2—12 noon at Norfolk County Hospital. Stated meeting. Speaker: Dr. Timothy Leary. Subject: Comparative Experimental Arteriosclerosis with Human Coronary Sclerosis.

December 7—12 noon at Quincy City Hospital. Program by the hospital staff.

January 4—12 noon at Norfolk County Hospital. Speaker to be announced.

February 1—12 noon at Norfolk County Hospital. Stated meeting. Speaker: Dr. Sara Jordan. Subject: Stomach Ulcers.

March 1—12 noon at Quincy City Hospital. Program by the hospital staff.

April 5—12 noon at Norfolk County Hospital. Speaker: Dr. Elliott P. Joslin. Subject: Diabetes.

May 3—12 noon at Norfolk County Hospital. Annual Meeting. Election of Officers.

BOOK REVIEWS

The Surgical Clinics of North America. Volume 13, Number 4. August, 1933. Mayo Clinic Number. W. B. Saunders Company, Philadelphia.

From the great wealth of material of the Mayo Clinic the staff have selected many of their most interesting cases for presentation in this volume. In general this issue consists principally of comparatively brief reports of a large number of unusual conditions. Some of the topics receiving more thorough discussion are: "Hypertrophic Stenosis of the Pylorus in Adults" by Judd and Thompson; "Chronic Duodenal Obstruction" by Judd and Puestow; "Retroperitoneal Tumors" by Judd and Larson; "Neurosurgical Treatment of Muscular Spasms and Spastic Painful and Trophic Lesions of the Extremities" by Adson; and "Renal Tuberculosis" by Droegemueller.

As a whole this volume will be found of greatest value in allowing the average surgeon to familiarize himself with many conditions which he will see but rarely.

The Control of Football Injuries. By MARVIN A. STEVENS, M.D., and WINTHROP M. PHELPS, M.D. A. S. Barnes & Company, New York City. 231 Pages. Price $3.00.

In this book the authors have arranged a clear and concise exposition on the fundamental principles of the popular American game of football. The illustrations on the technique of the game are excellent and admirably demonstrate the points brought out in the text. This type of book has been sorely needed and it is hoped that it will influence a reduction in the incidence of injury in this sport.

From the medical point of view this book covers the more common types of injury. Had it been designed solely for the medical press, it should have been more inclusive and better illustrated in this section. However, it contains certain valuable information for coaches, trainers, and team physicians.

Surgical Pathology. By WILLIAM BOYD, M.D. W. B. Saunders Company, Philadelphia, 1933. Pp. 866. Price $10.00.

The appearance of a third edition of Boyd's Surgical Pathology within eight years attests its popularity. Dr. Boyd's lucid and pleasing style, together with his catholic yet discriminating presentation, make an unusually useful volume for reference and for textbook purposes. Dr. Boyd's own wide experience enables him to weigh divergent theories and controversial points.

The numerous illustrations are well selected and a fair balance is kept between presentation of gross and microscopic pathological material. This volume, of course, presents some overlapping of the Pathology of Internal Diseases and the Textbook of Pathology by the same author. For the general practitioner, the surgeon, or the pathologist this volume is undoubtedly the most useful of the three.